Dr. Carol Rivers'

PREPARING FOR THE WRITTEN BOARD EXAM IN EMERGENCY MEDICINE

Seventh Edition

Volume I

Cardiovascular Disorders
Head, Ear, Eye, Nose, and Throat Disorders
Abdominal and Gastrointestinal Disorders
Thoracic and Respiratory Disorders
Traumatic Disorders
Orthopedic Emergencies
Musculoskeletal Disorders (Nontraumatic)
Nervous System Disorders
Gynecologic and Obstetric Disorders

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Ohio ACEP
American College of Emergency Physicians
Advocacy | Education | Leadership

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Published by:

Ohio Chapter, American College of Emergency Physicians (Ohio ACEP)
3510 Snouffer Road
Suite 100
Columbus, Ohio 43235

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www.ohacep.org

Printed in the United States of America

Seventh Edition

ISBN 978-0-9843856-8-3

Date of original release: January 1, 1992
Most recent update: February 24 , 2015

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The object of education is not learning, but discipline and enlightenment of the mind.

Woodrow Wilson

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FOREWORD

Welcome to the seventh edition of the *Dr. Carol Rivers' Preparing for the Written Board Exam in Emergency Medicine*. Initially created by Dr. Carol S. Rivers, a national leader in board review education, the Rivers "Blue Books" have long been the gold standard for comprehensive emergency medicine review and exam preparation. Dr. Rivers was residency trained in emergency medicine and extensively involved in the educational aspects of emergency medicine as a lecturer, a writer, a featured speaker, instructor, and tutor...helping those who wanted and needed a firmer grasp of the practice of emergency medicine. In 1983, Dr. Rivers began to focus her teaching efforts on preparing physicians for the certifying examinations in emergency medicine.

We celebrate her passion as a teacher, role model, and mentor. Since her early days as a leader and visionary in the specialty of emergency medicine, Dr. Rivers devoted her life to the advancement of emergency medicine and to the improvement of patient care, one physician at a time. Through her educational endeavors, providing publications and tutorials for emergency physicians and residents, she demonstrated outstanding service, leadership, and commitment to the specialty of emergency medicine. Ohio ACEP is proud to continue the tradition of the Rivers' board review with this current revision.

This book was envisioned, written, and compiled to fill a need—the need for a teaching textbook (not another reference book) aimed directly at the written board examination (not the entire field of emergency medicine) and designed for the busy physician (not the average medical student) who has limited time to devote to study and whose study time comes in unequal segments.

In its seventh edition, *Dr. Carol Rivers' Preparing for the Written Board Exam* continues the tradition as a premier teaching textbook for preparing for the Written Board exam in emergency medicine. Significant improvements have been made to this text to enhance the reader's experience and to cover the core content of the "Model of the Clinical Practice of Emergency Medicine" (The EM Model) and with consideration for the "Table of Specificity" of the American Board of Osteopathic Board of Emergency Medicine. We are confident that your use of this two-volume review text will give you a foundation for exam preparation and for passing an emergency medicine written certification exam.

INTRODUCTION

This book has been written primarily for emergency physicians preparing for a written certification or recertification exam, and for residents preparing for in-service exams in emergency medicine. Its purpose is to provide a concise, focused review of material that usually appears on these exams. Information that may seem "basic" is included, because it is essential to your understanding of principles and concepts that are important parts of the exam.

While this book has been used as a reference text, it is primarily a teaching text. The largest division of the book—the review of medical topics—is essentially a set of notes that contain the knowledge you must have to pass the exam. Treat them as notes—as your own notes. Read them with a pen, pencil, or highlighter in hand. Underline or highlight information you especially want to remember. Write notes of your own in the margins and other space provided. As you read, you will notice that some material is repeated—on purpose. A teaching text requires some redundancy for emphasis and coherence.

The seventh edition is an expanded and updated revision of the prior editions. A few new topics have been added that are pertinent to both the board exam and the residency in-service exams. Outdated information has been removed, and controversial issues have been clarified wherever possible. To enhance clinical acuity for the resident in training, a few bedside diagnostic techniques, in addition to descriptions of selected procedures, are included. The remaining text is essentially unchanged except for those changes required to bring the content in line with the EM Model.

You will occasionally be referred to other materials to enhance your study. Depending on how much time you have to prepare for this exam (see "Recommended Study Plan"), you may find the following references helpful:

1. Tintinalli, et al, *Emergency Medicine: A Comprehensive Study Guide,* Seventh ed. McGraw-Hill Company, Inc. (referred to in this text as Tintinalli's text).
2. Marx, et al, *Rosen's Emergency Medicine: Concepts and Clinical Practice,* Eighth ed., 2 vols. Saunders (referred to in this text as Rosen's Text).
3. *Physician's Evaluation and Educational Review in Emergency Medicine (PEER)* content review and self-assessment products (ACEP).
4. The bookstore at Ohio ACEP (www.ohacep.org)

These are supplemental resources. If asked to review a particular subject (eg, LeFort facial fractures), you should go to Tintinalli's and/ or Rosen's texts and read the appropriate section. Another text that has become popular with practicing emergency physicians is *Hardwood-Nuss' Clinical Practice of Emergency Medicine* (Lippincott Williams & Wilkins, Philadelphia). In general, it is easier to read than Tintinalli or Rosen and uses a numbered, itemized format similar to that found in this text.

The *PEER* self-assessment products may be valuable in your preparation, because they reacquaint you with the process of answering multiple-choice questions and help you define areas of weakness that require further study.

Several radiographic images are on the written exam. Whenever possible, those that merit review are noted. As you read through the medical topics included in this text, it is recommended that you have a pad of note paper and a pen handy. As you come to a paragraph in which a radiograph or CT review of a particular entity is recommended, jot it down. When you have completed your list, review common radiographs for each diagnosis. This is important for you to do because the radiographs, CT, and MRI prints on the written board exam are not usually taken from standard textbooks; they usually come from teaching files. A good text reference is *The Radiology of*

Emergency Medicine by Harris and Harris. You can also view radiographs, CTs, and MRIs online with Ohio ACEP's case pictorial review at www.ohacep-elearning.org.

ECGs and other pictorials are also presented on the exam. An ECG text that has been recommended is *ECGs for the Emergency Physician* by Mattu and Brady (Blackwell). Board preparation courses traditionally have hundreds of photos and ECGs that are ideal for many participants. If a course is not feasible, Ohio ACEP has published a series of books called *Photo and X-ray Stimuli for Emergency Medicine*. These texts contain more than 200 high-quality photos accompanied by clinical presentations. Additionally, Ohio ACEP's Web-based e-learning site features over 1,4000 cases that include diagnostic photos, radiographs, ultrasounds, and other images integrated with key facts to reinforce key concepts of emergency medicine. Another excellent pictorial source is *Atlas of Emergency Medicine* by Knoop, Stack, and Storrow (McGraw-Hill). In addition to clinical presentations, each entity includes a differential diagnosis, emergency department treatment and disposition, and clinical pearls.

A special feature of this text is clinical presentations of specific diagnoses as clinical scenarios in a storytelling format. This concept is based on the "simple chain technique," the object of which is to "chain" or link one item to the next in the order you wish to remember them. Basically, this chain is a story that involves all the items you want to remember in a particular sequence. You will be able to do this by using mental images and tying each item to the next. How does the chaining technique work? One item acts as a stimulus (or cue) for the next item. It's almost like seeing a slide show or a movie in your mind's eye, where you can automatically anticipate the next scene. You don't have to strain your mental faculty searching for it; it's right there.

Chaining has been tested in scientific studies that have verified its effectiveness as a memory-enhancing tool for simple rote memory tasks. In this respect, imagery is probably the single most important aspect of memory training. In numerous studies, one group of students would be shown pictures of an array of items to remember, and the other group would simply be given a list of words to remember. Both groups would be given the same test to determine what they remembered. The people who had been given images consistently scored higher than those who had been given lists. The important point here is that vivid images of items will improve your memory of those items.

Why should the seemingly insignificant procedure of linking one item to another dramatically improve recall? The answer is found to be in the linking itself. Separate pieces of information become unified when connected to each other. In other words, when 20 pieces of information are presented separately, you have to remember 20 independent segments of information. But when we link the items together, as we do in chaining, the 20 items actually represent only one segment of data.

Some areas of this text do contain memory recall facts (ie, lists), and they are unavoidable. However, these "lists" are easier to remember when they are mixed with imagery. Pure memorizing is a left brain function. Imagery is a right brain function. When combined, the reader remembers more detail.

Before you begin reading this text, briefly scan each of the sections so that you can get an overview of the information presented. You might want to read the nonclinical/nonacademic sections first, because other activities are discussed there and you will want to allow time to schedule some or all of them in the time before the exam. You have enough information in this text to pass the exam, but other activities can help you as well. So pick and choose those that sound worthwhile, and then plan your time to include them in your final preparation.

RECOMMENDED STUDY PLAN

Engage in regular and consistent study, because knowledge is acquired by studying over a reasonable period of time. What is learned in a hurry is seldom completely learned and soon forgotten. Do not "cram" for this exam. Cramming is an attempt to learn in a very short period of time what should have been learned through regular and consistent study over a period of weeks or months. Cramming seldom pays off in terms of effective learning. If you cram, it is likely that you will be more confused than prepared on the day of the exam.

Conditions and Components

Before you start reading this text, an organized approach will help maximize your time and effort. It is important to strategize and focus on the topics below that generally have a greater degree of emphasis on the exams.

Traumatic Disorders	11%
Cardiovascular Disorders	10%
Signs, Symptoms, and Presentations	9%
Abdominal and Gastrointestinal Disorders	9%
Thoracic-Respiratory Disorders	8%
Procedures and Skills	6%
Head, Ear, Eye, Nose, and Throat Disorders	5%
Systemic Infectious Disorders	5%
Nervous System Disorders	5%
Obstetrics and Gynecology	4%
Toxicologic Disorders	4%
Endocrine, Metabolic and Nutritional Disorders	3%
Environmental Disorders	3%
Musculoskeletal Disorders (Nontraumatic)	3%
Psychobehavioral Disorders	3%
Renal and Urogenital Disorders	3%
Other Core Components	3%
Cutaneous Disorders	2%
Hematologic Disorders	2%
Immune System Disorders	2%
Total	**100%**

Pediatric: 8% minimum
Geriatric: 4% minimum

The next point you need to consider is how much time you have left before the exam. Check off the time frame that is most appropriate to your situation.

__ Less than one month
__ One to two months
__ Two to three months
__ Three months or more

Now that you know which topics are covered most heavily on the written board exam and how much time you have left to study, a methodical approach is in order. There are four steps in this process:

1. A self-assessment evaluation to determine strong and weak areas.
2. A comparison of your self-assessment score with an analysis of your current level of preparedness for the exam.
3. A study plan that includes the number of hours per day and the number of days per week you need to study.
4. A method of reading and reviewing that promotes high retention and recall of specific information.

These four points will be described further in the paragraphs that follow. Using the time frame that you previously selected, choose the category that pertains to you and read the program outlined therein. That is your program, your study-approach method. Some of you will already have a plan in mind and may decide to incorporate parts of the information from within the different categories, and that's fine. But for those looking for direction, these categories have been written primarily for you.

Category I: Less than 1 month

1. Preceding each academic chapter is a series of multiple-choice questions. Go through the questions for every chapter in the academic section, recording your answers in the book or on a separate sheet of paper. When you're finished, use the answer key at the end of each series of questions and mark your incorrect answers. Use the pre-chapter multiple-choice question worksheet (on page xx) to record and determine your percentage of correct answers for each section.
2. Analyze your percentage score for the questions from each chapter to see where you are in the scheme of things:
   a. 85% or higher is a good score. Review the questions missed, and note the correct answers.
   b. 75%–85% is reasonably good, but further study is needed. For the questions you missed, review those particular subjects within the body of that chapter.
   c. Anything <75% is not a good score. Read the entire chapter, and then go through all the questions for that chapter again. If you have still missed some questions, review those subjects again.
   d. With this approach in mind, start at the top of the list of components provided above, and work your way down. That way you'll cover the most important topics first.

3. Plan your study schedule. Because you have less than a month, you'll need to study about 4 hours a day, 6 days a week. It is recommended that you study in two 2-hour blocks rather than 4 hours in one sitting. Your retention will be better, and you'll have more stamina.

4. If you have to read a whole chapter or parts of a chapter, do so with a marker in hand, highlighting important points. **The material most likely to be tested already appears in bold type.** If you are short on time, read only the bolded parts of each chapter. Plan to review this material three times. If you run out of time and can't finish this program, let it go and don't worry about it. You will have covered the most important material in the time available, and your chance of passing will be higher than if you tried to cram.

5. A few days before the exam, review all the pre-chapter questions and practice clinical scenarios again. Then go through the Critical Q & A's and questions (look up the answers if you have to). Do not stay up late the night before the exam. You will have better recall ability during the test if you are not tired.

Category II (1–2 months)

1. Preceding each academic chapter is a series of multiple-choice questions. Go through the questions for every chapter in the academic section, recording your answers in the book or on a separate sheet of paper. When you're finished, use the answer key at the end of each series of questions and mark your incorrect answers. Use the prechapter multiple choice question worksheet (on page xx) to record and determine your percentage of correct answers for each section.

2. Analyze your percentage score for the questions from each chapter to see where you are in the scheme of things:

   a. 85% or higher is a good score. Review the questions missed, and note the correct answers.
   b. 75%–85% is reasonably good, but further study is needed. For the questions you missed, review those particular subjects within the body of that chapter.
   c. Anything <75% is not a good score. Read the entire chapter, and then go through all the questions for that chapter again. If you have still missed some questions, review those subjects again.
   d. With this approach in mind, start at the top of the list of components provided above, and work your way down. That way you'll cover the most important topics first.

3. Plan your study schedule. In this time frame, you will need to study about 4 hours a day, five days a week. It is recommended that you study in two 2-hour blocks rather than 4 hours at one sitting. Your retention will be better, and you'll have more stamina.

4. As you go through the academic chapters (either in whole or in part), read with a marker in hand, highlighting important points. When you have finished, go back and reread the material you covered three more times. **The material most likely to be tested already appears in bold type.** If you are short on time, read only the bolded parts of each chapter. Plan to review this material three times. Be sure to review the radiographs and other pictorials noted in the text, using the recommended sources. If you still have some time left before the exam, go through the academic chapters that you have not reviewed and look for "pearls" (information that looks important) and other information that clarifies your understanding of a particular subject; review these points two or three more times and then stop. You're in reasonably good shape. Relax.

5. A week before the exam, go through the Critical Q & A's and the practice clinical scenarios.

Category III (2–3 months)

1. With additional time to prepare, a *PEER* content review and self-assessment testing product published by ACEP (available in the ACEP online bookstore) may be of value to your study plan. The *PEER* instructional model is composed of self-assessment study and practice, post-test, and evaluation. You may complete the online pretest, which will give you an overall and per category score. These results will give you a good sense of your priority study areas. Compare your score with the following analysis to see where you are in the scheme of things.

   a. 85% or higher is a good score. Review the questions missed, note the correct answers, and read the explanations given in the *PEER* product answer sections.

   b. 75%–85% is a reasonably good score, but further study is needed. In addition to reading the explanations to incorrect answers, also read the referenced material in Tintinalli's text.

   c. Anything <75% is not a good score. More extensive study is necessary, especially if several of the missed questions refer to material in the first nine topics on the list of components provided earlier. These components account for up to 68% of the material covered on the exam. You should read corresponding chapters in Tintinalli's text (as well as in this text). For the remaining chapters, it will be sufficient to read these in their entirety in this book; no other reference is necessary.

2. Plan your study schedule. If you have about 2 months, set aside about 3 hours a day, 5 days a week; it is recommended that you do not study more than 2 hours at one sitting, because your ability to retain information is likely to diminish after that time. If you have 3 months to study, 2 hours a day, 5 days a week should be sufficient.

3. As you go through this text, use a marker to highlight key points, ie, information you didn't know previously or that seems important. Be sure to review the radiographs and other pictorials noted in the text, using the recommended sources. Divide your material into "easy reading" and "hard reading" categories. An example might be:

Easy Reading	Hard Reading
Mechanics of the Written Board Exam	*PEER* test-taking and review
"Light" academic chapters (eg, cutaneous, EMS, emergency department administration)	The first nine academic chapters (especially Cardiovascular Disorders)
Additional Tips for Good Performance	Tintinalli's text reading

   Alternate your study time between the two categories. Do "hard reading" during one study session and "easy reading" the next. This will maximize your learning time as well as allow you to maintain your stamina. When you have finished, go back and reread this entire review book, looking especially for "pearls" (information that looks important) and other information that clarifies your understanding of a particular subject. Be sure to go through the questions preceding each academic chapter and record your scores on page xx; it will help focus your attention as you read the academic material. If you have time, reread again but, this time only the material you have highlighted, and then stop. **The material most likely to be tested already appears in bold type.**

4. In the final 7–10 days before the exam, go through the Critical Q & A's and practice clinical scenarios two or three times and stop studying the day before the exam. You're ready.

Category IV (3 months or more)

1. With additional time to prepare, a *PEER* content review and self-assessment testing product published by ACEP (available in the ACEP online bookstore) may be of value to your study plan. The *PEER* instructional model is composed of self-assessment study and practice, post-test, and evaluation. You may complete the online pretest, which will give you an overall and per category score. These results will give you a good sense of your priority study areas. Compare your score with the following analysis to see where you are in the scheme of things.
   a. 85% or higher is a good score. Review the questions missed, note the correct answers, and read the explanations given in the *PEER* product answer sections.
   b. 75%–85% is a reasonably good score, but further study is needed. In addition to reading the explanations to incorrect answers, also read the referenced material in Tintinalli's text.
   c. Anything <75% is not a good score. More extensive study is necessary, especially if several of the missed questions refer to material found in the first nine topics on the list of components provided above; these topics represent up to 68% of the material covered on the exam. You should read corresponding chapters in Tintinalli's text (as well as in this text). Of the remaining chapters, it will be sufficient to read these in their entirety in this book; no other reference is necessary.
2. Plan your study schedule. If you have 3–4 months before the exam, plan on studying about 2 hours a day, 5 days a week. If you have more than 4 months, 2 hours a day, 4 days a week is sufficient. You may also want to attend a written board course. (See the section titled "Additional Tips for Good Performance" for info on written board courses).
3. As you go through this text, use a marker to highlight key points, ie, info you didn't know previously or that seems important. The material most likely to be tested already appears in bold type. Be sure to review radiographs and other pictorials noted in the text, using the recommended sources. Divide your material into "easy reading" and "hard reading" categories. An example might be as follows:

Easy Reading	Hard Reading
Mechanics of the Written Board Exam	*PEER* test-taking and review
"Light" academic chapters (eg, cutaneous, EMS, emergency department administration)	The first nine academic chapters (especially Cardiovascular Disorders)
Additional Tips for Good Performance	Tintinalli's text reading

   Alternate your study time between the two categories. Do "hard reading" during one study session and "easy reading" the next. This will maximize your learning time as well as allow you to maintain your stamina. When you have finished, go back and reread the entire book, looking especially for "pearls" (info that looks important) and any information that clarifies your understanding of a particular subject. Be sure to go through the questions preceding each academic chapter and record your scores on the next page; it will help focus your attention as you read the academic material. If you have time, reread again but, this time, only material you have highlighted, and then stop.
4. In the last 2 weeks before the exam, go through the Critical Q & A's and practice clinical scenarios until you know all the answers. Then stop studying. You are well prepared.

Regardless of in which "category" you place yourself, test-taking skills and strategies can play a significant role in your exam score. Be sure to read "How To Take a Multiple-Choice Exam" in the section titled "Mechanics of the Written Board Exam." Practice using the techniques described

for answering questions for which you don't know the answers. Start first with the questions in this book. If you have time, do the same with the *PEER* self-assessment testing products. Use other sources as well, such as the Critical Q & A's that supplement this text). Some written board preparation courses also offer practice questions. Under no circumstances is it recommended that you focus your attention on test-taking techniques at the expense of studying the academics of emergency medicine. If your knowledge base is lacking, no amount of test-taking skill is going to result in a passing score. In addition, these techniques don't work all the time. But if you're one of those candidates who scores 73%–74% on exam content, knowing how to use test-taking techniques can raise your score another percent or two— just enough to help you pass.

Pre-chapter Multiple Choice Self-Assessment Questions Worksheet

Chapter	# answered correctly	# of pre-chapter questions	Percentage of correct answers	Missed questions to review
Cardiovascular Disorders		35		
Head, Ear, Eye, Nose, Throat Disorders		56		
Abdominal and Gastrointestinal Disorders		39		
Thoracic and Respiratory Disorders		27		
Traumatic Disorders		30		
Orthopedic Emergencies		35		
Musculoskeletal Disorders (Nontraumatic)		11		
Nervous System Disorders		16		
Gynecologic and Obstetric Disorders		19		
Pediatric Emergencies		41		
Toxicologic Disorders		21		
Endocrine, Metabolic, and Nutritional Disorders		29		
Environmental Disorders		34		
Psychobehavioral Disorders		15		
Hematologic Disorders		15		
Oncologic Disorders		11		
Systemic Infectious Disorders		14		
Immune System Disorders		14		
Renal and Urologic Disorders		11		
Cutaneous Disorders		17		
Emergency Medical Services		9		
Procedures and Skills		13		
Other Core Competencies		23		
Total		**535**		

Scoring

- 85% or higher is a good score. Review any questions missed, and note the correct answers.
- 75%–85% is reasonably good, but further study is needed. For the questions you missed, review those particular subjects within the body of that chapter.
- Anything <75% is not a good score. Read the entire chapter and then go through all the questions again. If you have still missed some questions, review those subjects again.

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CARDIOVASCULAR DISORDERS: SELF-ASSESSMENT QUESTIONS

1. ECG findings of tall T waves, wide QRS complexes, flattened P waves, and a prolonged PR interval are most consistent with the presence of:

   (a) Hypercalcemia
   (b) Hyperkalemia
   (c) Hypocalcemia
   (d) Hypokalemia

2. The presence on ECG of sagging ST segments, short QT intervals, and flattened or inverted T waves is most accurately described as:

   (a) Digitalis effect
   (b) Digitalis toxicity
   (c) Hypercalcemia
   (d) Hypokalemia

3. The optimal treatment of ventricular tachycardia and supraventricular tachycardia is:

   (a) Lidocaine
   (b) Adenosine
   (c) Cardioversion
   (d) Dependent on the hemodynamic stability of the patient

4. All of the following are indications for pacemaker therapy except:

   (a) Asymptomatic bifascicular or trifascicular block
   (b) Hemodynamically unstable bradycardia unresponsive to drug therapy
   (c) Mobitz II second-degree AV block in the presence of an acute MI
   (d) Overdrive of tachydysrhythmias refractory to drug therapy or electrical cardioversion

5. The most common cause of failure to pace is:

   (a) Battery depletion
   (b) Oversensing
   (c) Undersensing
   (d) Wire fracture

6. ST segment elevation in leads V_1–V_6 with reciprocal ST depression in leads II, III, and aVF is characteristic of:

   (a) Acute anterior wall MI
   (b) Acute inferior wall MI
   (c) Acute lateral wall MI
   (d) Acute posterior wall MI

7. All of the following drugs may be given through an endotracheal tube except:

   (a) Atropine
   (b) Epinephrine
   (c) Naloxone
   (d) Sodium bicarbonate

8. Appropriate immediate therapy for acute pulmonary edema may include all of the following agents except:

   (a) Digitalis
   (b) Furosemide
   (c) Nitroglycerin
   (d) Oxygen

9. What is the earliest radiographic finding of congestive heart failure?

   (a) Alveolar edema
   (b) Cardiomegaly
   (c) Interstitial edema
   (d) Pulmonary vascular redistribution to the upper lung fields

10. What is the most effective medical therapy for the treatment of atypical chest pain associated with hypertrophic cardiomyopathy?

    (a) ß-blockers
    (b) Digitalis
    (c) Morphine
    (d) Nitroglycerin

11. The diagnostic procedure of choice for detecting a pericardial effusion occurring in association with pericarditis is:

    (a) Chest radiograph
    (b) ECG
    (c) Echocardiography
    (d) Radionuclide scanning

12. The differential diagnosis of neck vein distention associated with hypotension includes all of the following except:

    (a) Acute mitral regurgitation
    (b) Infective endocarditis
    (c) Pericardial tamponade
    (d) Tension pneumothorax

13. Most pulmonary emboli originate from venous thrombi in the:

    (a) Calf
    (b) Heart
    (c) Lower extremities and pelvis
    (d) Upper extremities

14. All of the following statements regarding the use of aortography in the evaluation of acute aortic dissections are true except:

    (a) It can miss dissection if the false lumen is thrombosed.
    (b) It has been the traditional diagnostic gold standard.
    (c) It is 100% accurate.
    (d) Unlike rapid-sequence CT scanning with contrast, aortography also assesses the aortic valves and branches.

15. All of the following statements regarding DeBakey type III aortic dissections are accurate except:

    (a) Initial management of these dissections is medical.
    (b) They are equivalent to Stanford type B dissections.
    (c) They are the most common type of dissection.
    (d) Long-term management of these dissections is usually medical.

16. Medical therapy for aortic dissections is aimed at controlling the forces that propagate the dissection. The first-line agent(s) used to accomplish this goal is/are:

    (a) ß-blockers and nitroglycerin
    (b) ß-blockers and nitroprusside
    (c) Nitroprusside alone
    (d) Trimethaphan

17. A 30-year-old woman presents with palpitations of sudden onset and a feeling of nervousness. The patient denies drug use of any type and has had no other episodes of palpitations or anxiety. The nurse hands you a rhythm strip demonstrating an irregular wide complex tachycardia (QRS = 0.13 seconds) at 250 beats per minute. There is no evidence of torsades. The likely etiology of this rhythm is:

    (a) Anxiety
    (b) Cocaine use
    (c) Mitral valve prolapse
    (d) Wolff-Parkinson-White syndrome

18. Atypical chest pain associated with mitral valve prolapse is treated with:

    (a) ß-blockers
    (b) Morphine
    (c) Nitroglycerin
    (d) Oxygen

19. The treatment of choice for hypertension associated with eclampsia is:

(a) Diuretics
(b) Labetalol
(c) Magnesium sulfate and hydralazine
(d) Nitroprusside

20. The primary difference between a hypertensive urgency and a hypertensive emergency is that in a hypertensive emergency:

(a) It is usually treated with oral medications.
(b) The diastolic blood pressure is ≥130 mmHg.
(c) There is evidence of acute end-organ dysfunction or damage.
(d) There is no history of hypertension.

21. The ideal location of the catheter tip for temporary transvenous pacing is in the:

(a) Apex of the right ventricle
(b) Intraventricular septum
(c) Right atrium
(d) Superior vena cava

22. Successful placement of a temporary transvenous pacemaker under ECG guidance is indicated by the observation of _____ on the cardiac monitor.

(a) Small positive P waves and near-normal QRS complexes
(b) Prominent inverted P waves and smaller negative QRS complexes
(c) ST elevation
(d) ST depression

23. Placement of a pacemaker magnet over most permanent pacemakers results in:

(a) Conversion from a demand to a fixed-rate mode
(b) Conversion from a fixed-rate to a demand mode
(c) Permanent disabling of the pacemaker
(d) Temporary disabling of the pacemaker

24. A 31-year-old man is brought in by ambulance for evaluation of a syncopal episode that occurred while he was playing a friendly game of competitive basketball. Examination reveals a rapid biphasic carotid pulse and a prominent systolic ejection murmur along the left sternal border and at the apex. You suspect hypertrophic cardiomyopathy and ask the patient to perform the Valsalva maneuver while you auscultate his heart. Assuming your diagnosis is correct, you would expect the intensity of the murmur to _____ with this maneuver.

(a) Decrease
(b) Disappear
(c) Increase
(d) Remain unchanged

25. All of the following statements regarding hypertrophic cardiomyopathy are accurate except:

    (a) Amiodarone is the treatment of choice for the ventricular dysrhythmias that occur in patients with this disorder.
    (b) Digitalis and vasodilators are typically the most useful agents in the management of this disorder.
    (c) It is inherited in >50% of patients.
    (d) It is characterized by left septal hypertrophy without associated ventricular dilatation.

26. Antibiotic prophylaxis for bacterial endocarditis is definitely recommended in high-risk patients for which of the following procedures?

    (a) Endotracheal intubation
    (b) Dental procedures associated with significant gingival bleeding
    (c) Placement of a Foley catheter
    (d) Suturing of a laceration

27. Antibiotic prophylaxis for endocarditis is indicated in patients with all of the following conditions except:

    (a) Congenital heart disease
    (b) History of bacterial endocarditis
    (c) Mitral valve prolapse
    (d) Prosthetic heart valves

28. All of the following statements regarding prosthetic heart valves are accurate except:

    (a) Endocarditis should be suspected in any patient with a prosthetic valve who presents with fever, especially if a new regurgitation heart murmur is heard.
    (b) Mechanical valves require lifelong systemic anticoagulation.
    (c) Patients with valve dysfunction secondary to thrombus formation usually present with acute onset of CHF, hypotension, and muting (or loss) of the prosthetic valve sound.
    (d) Tissue valves cause greater hemolysis and are more thrombogenic than mechanical valves.

29. In patients with automatic implantable cardioverter defibrillators (AICDs) in place who require CPR:

    (a) CPR is performed in the usual manner.
    (b) The provider may perceive an AICD shock if the device has not been deactivated.
    (c) Perception of an AICD shock by a provider is neither dangerous nor uncomfortable.
    (d) All of the above are correct.

30. Janeway lesions are most accurately described as:

    (a) Nontender, erythematous macular lesions on the fingers, palms, soles
    (b) Nontender erythematous nodules on the dorsal aspect of the fingertips
    (c) Retinal hemorrhages with central clearing
    (d) Tender, erythematous nodules on the volar aspect of the fingertips

31. The most useful test for making the diagnosis of endocarditis is:
    (a) Blood culture
    (b) CBC
    (c) ECG
    (d) Erythrocyte sedimentation rate

32. The organism responsible for most cases of right-sided endocarditis is:
    (a) *Enterococcus* spp
    (b) *Streptococcus epidermidis*
    (c) *Streptococcus viridans*
    (d) *Staphylococcus aureus*

33. Which of the following statements regarding mitral stenosis is false?
    (a) Typical presenting symptoms include dyspnea on exertion and hemoptysis.
    (b) Common ECG findings are left atrial enlargement and atrial fibrillation.
    (c) Most cases are the result of rheumatic heart disease.
    (d) The most common complication is infective endocarditis.

34. The type of CHF that occurs in association with beriberi is most accurately characterized as:
    (a) High-output left ventricular failure
    (b) High-output right ventricular failure
    (c) Low-output left ventricular failure
    (d) Low-output right ventricular failure

35. When compared with the ST elevation that occurs in association with acute MI, the ST elevation that occurs in association with acute pericarditis is:
    (a) Concave upward in configuration
    (b) More diffuse
    (c) Nonanatomic in distribution
    (d) All of the above

ANSWERS

1. b	8. a	15. c	22. c	29. d
2. a	9. d	16. b	23. a	30. a
3. d	10. a	17. d	24. c	31. a
4. a	11. c	18. a	25. b	32. d
5. b	12. b	19. c	26. b	33. d
6. a	13. c	20. c	27. c	34. a
7. d	14. c	21. a	28. d	35. d

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

DYSRHYTHMIAS

I. BASIC PRINCIPLES OF CARDIAC CONDUCTION DISTURBANCES

A. Standard ECG and rhythm strips

1. Recordings are obtained at a paper speed of 25 mm/sec.
2. The vertical axis measures distance; the smallest divisions are 1 mm long ×1 mm.
3. The horizontal axis measures time; each small division is 0.04 sec/mm.

B. Normal morphology



Courtesy of Dr. Michael McCrea

1. P wave = atrial depolarization
   a. Upright in leads I, II, III, aVL, and aVF; inverted in lead aVR
   b. Measures <0.10 seconds wide and <3 mm high
   c. Normal PR interval is 0.12–0.20 seconds.
2. QRS complex = ventricular depolarization
   a. Measures 0.06-0.10 seconds wide
   b. Q wave is <0.04 seconds wide and <3 mm deep; the Q wave is abnormal if it is >3 mm deep or >1/3 of the QRS complex.
   c. R wave ≤7.5 mm high
3. QT interval varies with rate and sex but is usually 0.33–0.42 seconds; at normal heart rates, it is normally <½ the preceding RR interval.
4. T wave = ventricular repolarization
   a. Upright in leads I, II, V_3–V_6; inverted in aVR
   b. Slightly rounded and asymmetric in configuration
   c. Measures ≤5 mm high in limb leads and ≤10 mm high in the V leads
5. U wave = a ventricular afterpotential
   a. Any deflection after the T wave (usually low voltage)
   b. Same polarity as the T wave
   c. Most easily detected in lead V_3
   d. Can be a normal component of the ECG
   e. Prominent U waves may indicate one of the following:
      (1) Hypokalemia (<3 mEq/L)
      (2) Hypercalcemia

(3) Therapy with digitalis, phenothiazines, quinidine, epinephrine, inotropic agents, or amiodarone

(4) Thyrotoxicosis

f. Inverted (negative) U waves may indicate one of the following:

(1) Acute coronary ischemia

(2) Ventricular strain, dilation, or overload

(3) Hypertension

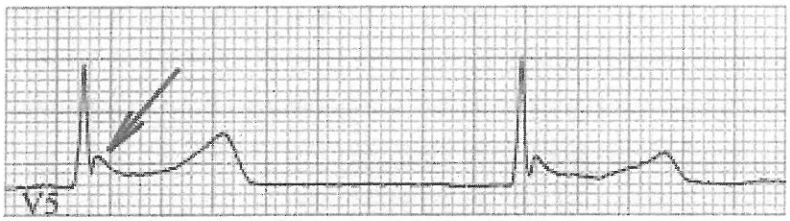
(4) Intracranial or subarachnoid hemorrhage

C. Causes of abnormal morphologies

1. Hypothermia: core temperature <95°F (35°C)

a. ECG findings

(1) "J wave" (also referred to as an "Osborn wave"): a broad, upright deflection at the end of an upright QRS complex



Courtesy of Dr. Michael McCrea

(2) Conduction delays: PR, QRS, and QT intervals may all be prolonged. QT interval is prolonged primarily because of selective prolongation of the ST segment.

(3) Dysrhythmias: sinus bradycardia and atrial fibrillation with a slow ventricular response are the most commonly encountered in this setting; the risk of developing dysrhythmias increases as the core temperature falls below 86°F (30°C); at core temperatures below 77°F (25°C), spontaneous ventricular fibrillation and asystole may occur. (These patients must be handled gently, because dysrhythmias are easily introduced).

b. Management of dysrhythmias

(1) Most dysrhythmias in hypothermic patients usually require only supportive therapy, because the dysrhythmias resolve spontaneously with rewarming.

(2) Cardiac arrest

(a) In profoundly hypothermic patients who appear to be in cardiac arrest, palpating pulses may be extremely difficult.

(b) If unsure, began CPR without delay.

(3) Ventricular fibrillation

(a) Often refractory to defibrillation attempts until the patient is rewarmed

(b) Defibrillation should be attempted up to three shocks but, if unsuccessful, CPR and rapid rewarming measures should be instituted. Further attempts at defibrillation should be withheld until the patient's temperature rises above 86°F (30°C).

(c) As the myocardium rewarms, ventricular fibrillation may convert spontaneously or in response to defibrillation.

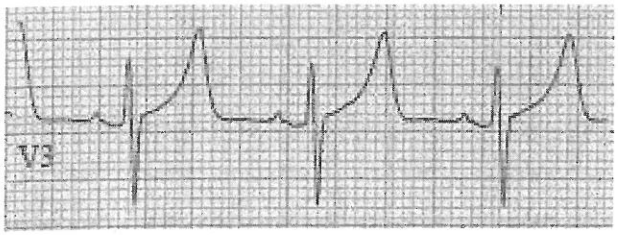
(d) Magnesium sulfate produces spontaneous conversion in these patients.

(4) The role of ACLS medications, including vasopressors, in severe hypothermic patients in cardiac arrest is of uncertain value, and standard algorithms may be used.

(5) If narcotic abuse is suspected, naloxone should be considered, because it may act on central opiate receptors to decrease the severity of hypothermia seen in overdoses.

(6) In general, a patient is not considered "dead" until "warm and dead," with warm being 95°F (35°C).

2. Hypokalemia
   a. Progressively more prominent U wave (best seen in V_3)
   b. Flattening of T wave (earlier) followed by inversion (later)
   c. Depression of ST segment
   d. Prominent P wave
   e. Prolongation of the PR and QT (U) interval
   f. Ventricular tachycardia/torsades
   g. In the presence of hypokalemia, susceptibility to digitalis toxicity and its associated dysrhythmias is increased.
3. Hyperkalemia



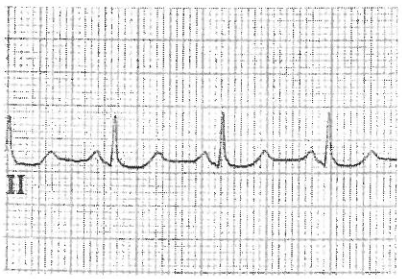
Courtesy of Dr. Michael McCrea

   a. Tall hyperacute T wave (earliest ECG finding)
   b. Prolonged PR interval
   c. Flattened or absent P wave
   d. Wide QRS complex that eventually blends with the T wave to assume a "sine wave" appearance
   e. Heart blocks
   f. QT interval normal or shortened

Table 1: Effects on ECG with Increasing Potassium Concentration

Potassium Concentration (mEq/L)	ECG Changes
5.5–6.5	PR interval prolongation P wave flattening/disappearance QRS widening Conduction block with escape beats
6.5–8.0	Slight pain
>8.0	Sine wave appearance Ventricular fibrillation Asystole

4. Hypocalcemia
   a. Prolonged QT interval because of prolongation of the ST segment
   b. Terminal T wave inversion (less consistent finding)
   c. Ventricular dysrhythmias (including torsades de pointes)
5. Hypercalcemia
   a. Depressed ST segments, widened T waves, and shortened ST segments and QT intervals.
   b. Bradyarrhythmias may occur, with bundle-branch patterns that may progress to second-degree block or complete heart block.
6. Hypomagnesemia
   a. Prolonged PR and QT intervals
   b. Widened QRS complex
   c. ST segment abnormalities
   d. Flattened or inverted T waves (especially in the precordial leads)
   e. Ventricular dysrhythmias (premature ventricular contractions, ventricular tachycardia, torsades de pointes, ventricular fibrillation)
   f. Hypomagnesemia usually occurs in association with other electrolyte abnormalities (particularly hypokalemia), and many of the ECG findings are similar to those seen with hypokalemia and hypocalcemia (pictured above).
   g. In the presence of hypomagnesemia, susceptibility to digitalis toxicity and its associated dysrhythmias is increased.
7. Digitalis effects



   a. Sagging ST segment with its concavity directed upward (resembles a hockey stick)
   b. Short QT interval
   c. Flattened or inverted T wave
   d. Modestly prolonged PR interval

e. These effects are especially prominent in the lateral leads and occur in most patients who are adequately digitalized; they are not an indication of digitalis toxicity.

8. Digitalis toxicity
   a. Pathophysiology: digitalis produces toxicity by
      (1) Poisoning the Na^+-K^+-ATPase pump → increased intracellular entry of Na^+ and Ca^{++} and egress of K^+ → increased excitability → ectopy and tachydysrhythmias
      (2) Increasing vagal tone and automaticity → decreased conduction in the AV node → bradydysrhythmias and AV blocks
   b. Factors that increase sensitivity to digitalis and predispose to toxicity
      (1) Electrolyte abnormalities (hypokalemia, hypomagnesemia, hypercalcemia, and hyperkalemia)
      (2) Hypoxia
      (3) Metabolic alkalosis
      (4) Increasing age
      (5) Presence of underlying cardiac disease (ischemia, CHF, congenital heart disease)
      (6) Presence of chronic underlying systemic illness (COPD, renal failure, hypothyroidism)
      (7) Drug interactions (quinidine, calcium channel blockers, erythromycin, amiodarone, captopril, and ibuprofen)
   c. ECG findings
      (1) Premature ventricular contractions (most common arrhythmia), often bigeminal and multiform: the most common digitalis-induced rhythm disturbance
      (2) Junctional tachycardia (common)
      (3) Sinus bradycardia
      (4) Sinus tachycardia
      (5) Sinoatrial and AV nodal blocks
      (6) Sinus arrest
      (7) Torsades de pointes
      (8) Ventricular tachycardia
      (9) Ventricular fibrillation
      (10) Atrial tachycardias with AV block are very specific but not pathognomonic for digitalis toxicity
      (11) Nonparoxysmal junctional tachycardia
      (12) Atrial fibrillation with a slow ventricular response, ie, AV dissociation
      (13) Bidirectional ventricular tachycardia (highly suggestive of digitalis toxicity but rare)
   d. Clinical symptoms
      (1) Flu-like syndrome with profound malaise, anorexia, nausea, vomiting, and diarrhea
      (2) Visual disturbances (blurred vision, halos around objects, and yellow or green color aberrations)
      (3) Mental status changes, including confusion, drowsiness, and psychosis
   e. Acute digitalis toxicity is usually seen in young and otherwise healthy patients as a result of either accidental or intentional overdose; it is commonly associated with hyperkalemia, high digoxin levels, bradydysrhythmias, and AV blocks. Toxicity in these patients is most closely correlated with the degree of hyperkalemia (not the serum digoxin level). Chronic digitalis toxicity generally occurs in older cardiac patients with

reduced renal function who are taking diuretics. These patients are usually normo- or hypokalemic, have digoxin levels that are minimally increased or normal, and most commonly have a ventricular dysrhythmia.

f. Classic clinical scenario

(1) Acute intoxication: A 3-year-old is brought in by his parents for evaluation after accidental ingestion of grandpa's "heart pills." Based on information obtained from the parents, the child has ingested 10.7 mg of digoxin sometime within the past 2 hours and has vomited twice. The cardiac monitor shows a junctional rhythm with sinus block and type I second-degree AV block; laboratory studies reveal a potassium level of 6.2 mEq/L, along with a markedly increased digoxin level of 61. The child is on no medications and is otherwise healthy.

(2) Chronic intoxication: A 65-year-old woman with a past medical history of coronary artery disease, CHF, and renal insufficiency is brought in by ambulance for evaluation. Her medications include furosemide, digitalis, sublingual nitroglycerin, and baby aspirin. According to family members, she has become progressively more confused and weak over the past few days and has not been eating well. The ECG shows a regular wide complex tachycardia with alternating QRS polarity (bidirectional ventricular tachycardia) and laboratory studies reveal a digoxin level of 3.5 and a potassium concentration of 3.0 mEq/L.

g. Management

(1) IV line, oxygen, pulse oximeter, cardiac monitor

(2) Gastric lavage is contraindicated because of risk of vagal stimulation causing bradycardia or asystole.

(3) Administer multiple doses of activated charcoal to all patients with potentially toxic ingestions; activated charcoal prevents systemic absorption and, when multiple doses are given, enhances elimination by interrupting the prominent enterohepatic circulation of digitalis.

(4) Seek and treat factors that may contribute to digitalis toxicity.

(a) Hypokalemia (correct cautiously in the presence of AV blocks; correction can actually exacerbate AV conduction defects)

(b) Hyperkalemia is best treated with Fab fragments (digoxin-specific antibody fragments); do not administer calcium; it can potentiate cardiotoxicity.

(c) Hypomagnesemia

(d) Hypoxia

(e) Dehydration

(5) Control tachydysrhythmias

(a) Phenytoin and lidocaine are the drugs of choice for tachydysrhythmias.

(b) Magnesium sulfate may also be useful in suppressing ventricular irritability.

(c) Avoid cardioversion (digoxin decreases the fibrillatory threshold); restrict its use to situations of last resort and use the lowest possible energy level.

(d) Avoid use of bretylium, Class IA antidysrhythmics (eg, procainamide, isoproterenol) and propranolol; these agents can exacerbate dysrhythmias and AV conduction disturbances.

(6) Manage symptomatic bradycardia or AV block with atropine. If atropine is unsuccessful, cardiac pacing (external or transvenous) may be used while waiting for Fab fragments to take effect. External pacing is preferred, because transvenous pacemaker insertion may induce tachydysrhythmias in these patients.

(7) Fab fragments
  (a) Should be administered to patients with:
    i. Ventricular dysrhythmias (ventricular fibrillation, ventricular tachycardia)
    ii. Symptomatic bradycardias unresponsive to atropine
    iii. Hyperkalemia (K^+ >5.0 mEq/L) secondary to digitalis intoxication
    iv. Coingestions of cardiotoxic drugs (ß-blockers, cyclic antidepressants)
    v. Large, potentially lethal digitalis intoxications
    vi. Ingestions of plants known to contain cardiac glycosides (oleander, lily of the valley) with severe dysrhythmias
  (b) Fab fragments bind free digoxin in the vascular and interstitial spaces and form an inert compound that is eliminated by the kidneys. Treatment rapidly corrects conduction defects, ventricular dysrhythmias, and hyperkalemia.
  (c) Dosage
    i. If the serum digoxin level or total amount of digoxin ingested is known, the formulas found in the package insert can be used to calculate the number of vials of Fab fragments to be administered.
    ii. If the amount of digoxin ingested is unknown, the initial dose of Fab fragments should be 5–10 vials (titrated incrementally).
  (d) After administration of Fab fragments, conventional assays for determining digoxin levels (which measure both bound and unbound digoxin) are unreliable for at least a week.

Table 2: ECG Findings in Various Conditions

Condition	ST	PR	QRS	QT	P wave	T wave	Special Features
Hypothermia		Long	Wide	Long			"J" wave, Osborne
Hypokalemia	Depressed	Long		Long		Flat	Progressively more prominent "U" wave
Hyperkalemia		Long	Wide		Flat	Peaked	May be associated with digitalis toxicity
Hypocalcemia				Long		Inverted	
Hypercalcemia				Short			
Hypomagnesemia	Abnormal	Long		Long		Flat	↑Susceptibility to digitalis toxicity
Digitalis effects	Scooped			Short		Flat	
Digitalis toxicity							Premature ventricular contractions most common

II. SPECIFIC RHYTHM ASSESSMENTS

A. Sinus rhythm



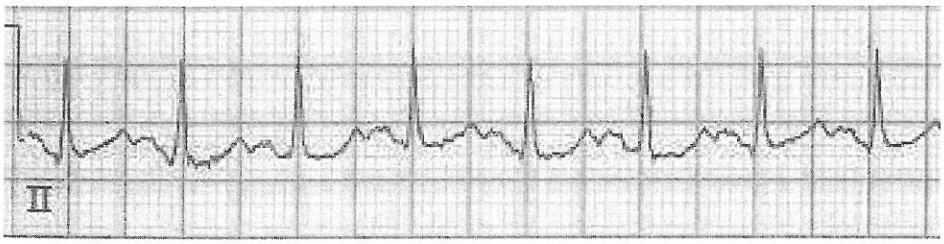
Courtesy of Dr. Michael McCrea

1. Sinus rhythm is 60–100 beats per minute.
2. The rhythm is regular with 1:1 relationship of the P to QRS.
3. P waves are upright in leads I, II, and aVF. (Lead II is the typical lead for a rhythm strip.)
4. There are no extra beats.

B. Premature atrial contractions: extra beats that originate outside the sinus node from ectopic atrial pacemakers; appear interspersed throughout an underlying rhythm (usually sinus)

1. These ectopic P waves are different in configuration from normal P waves and may or may not be conducted through the AV node. They usually have a normal PR interval (0.12–0.20 seconds)
2. They are generally followed by a noncompensatory pause; the SA node is reset, and the returning sinus beat occurs ahead of schedule.

C. Sinus tachycardia: exactly like a sinus rhythm except that the rate is >100 (and usually <160) beats per minute



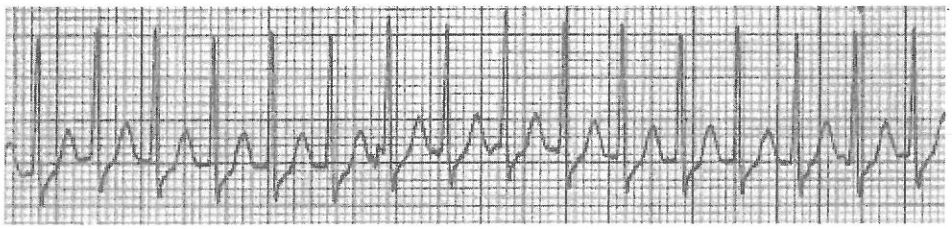
Courtesy of Dr. Michael McCrea

D. Sinus bradycardia: exactly like a sinus rhythm except that the rate is <60 (and usually >45) beats per minute



Courtesy of Dr. Michael McCrea

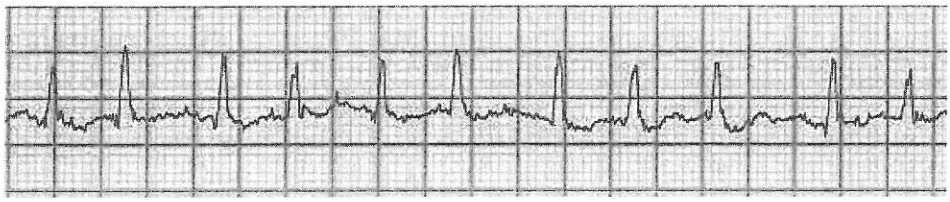
E. Supraventricular tachycardia



Courtesy of Dr. Daniel Schwerin

1. P waves are abnormal and may not be visible (often hidden in the preceding T wave), or they may immediately follow the QRS complex, in which case they are often inverted ("retrograde P waves"); atrial rate is 120–200 beats per minute.
2. Rhythm is regular.
3. QRS complexes are usually narrow but may be wide because of aberrant conduction through a bypass tract or preexisting bundle-branch block; ventricular rate is 120–220 beats per minute.

F. **Atrial fibrillation: an irregularly irregular rhythm due to uncoordinated atrial activation and random occurrence of ventricular depolarization. The atria are not pumping, but they do discharge electrical impulses to the ventricles; however, no single impulse depolarizes the atria completely, so only an occasional impulse gets through to the AV node. It is the most common sustained dysrhythmia; it occurs in 2% of the general population and in 5% of people >60 years old.**



Courtesy of Dr. Michael McCrea

1. P waves are absent but small irregular deflections in the baseline ("f" or "fibrillation waves") may be seen. They are most easily detected in the inferior leads (II, III, and aVF) and in V_1–V_3. The atrial rate is 400–700 beats per minute.
2. Because P waves are not visible, there is no PR interval.
3. QRS complexes are normal in configuration, unless there is aberrant conduction.
4. The rhythm is irregularly irregular.
5. Ventricular response rate is variable but is generally 160–180 beats per minute in untreated patients; a rate >200 beats per minute with a wide QRS complex suggests Wolff-Parkinson-White syndrome with antegrade conduction through the accessory pathway; a regular, slow ventricular rate may be digitalis toxicity.

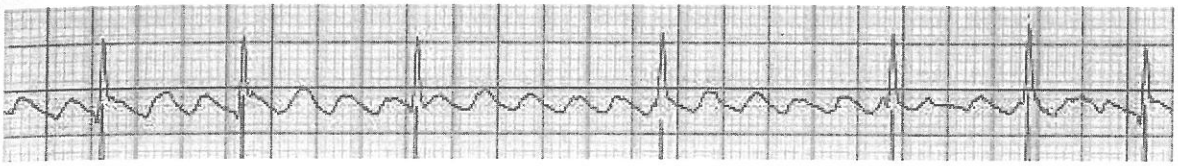
G. **Atrial flutter: a very rapid atrial rhythm but, because of AV nodal delay, ventricular responses are slower. Therefore, atrial flutter always occurs with some sort of AV block (not all impulses are conducted); the resulting block is either a fixed ratio (2:1, 3:1, 4:1, etc) or variable AV block.**

Fixed AV block 3:1 atrial flutter



Courtesy of Dr. Michael McCrea

Variable AV block atrial flutter



Courtesy of Dr. Michael McCrea

1. P waves have a characteristic sawtooth pattern and are called "F" or "flutter waves." They are usually best seen in the inferior leads and leads V_1 and V_2. The atrial rate is 250–350 beats per minute.
2. The PR interval (when present) is always normal, but not every P wave is followed by a QRS complex.
3. QRS complexes are normal in configuration.
4. The ventricular rate is often 100–150 beats per minute but depends on the degree of block present and may be variable. Suspect atrial flutter with a 2:1 block in patients who present with a fixed regular ventricular rate of 150 beats per minute.

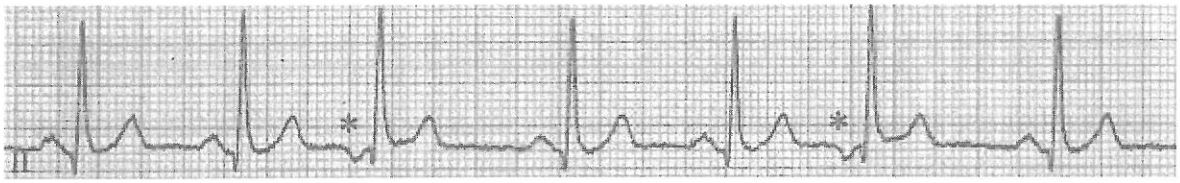
H. Multifocal atrial tachycardia: an irregular rhythm sometimes mistaken for atrial fibrillation; originates from multiple different atrial sites and is characterized by P waves of varying shape



Courtesy of Dr. Daniel Schwerin

1. There must be at least three distinct types of P waves in one lead; atrial rate is 100–180 beats per minute.
2. The rhythm is irregularly irregular.
3. The PP, PR, and RR intervals vary.
4. Nonconducted (blocked) P waves are frequently present, particularly when the atrial rate is rapid. Classically seen with COPD and theophylline toxicity. Management is directed at the underlying condition. Cardioversion is ineffective.

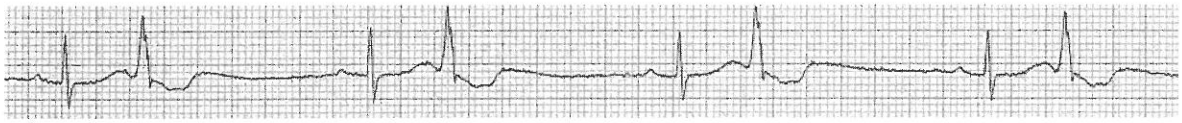
I. Junctional premature contractions: impulses that originate from an ectopic focus within the AV node or the bundle of His above the bifurcation. They may be isolated, multiple, or multifocal.



Courtesy of Dr. Michael McCrea

1. The ectopic P wave has a different shape and deflection (usually inverted in leads II, III, and aVF), and it may occur before, during, or after the QRS complex.
2. When the P wave precedes the QRS, the PR interval is shorter than normal (often <0.12 seconds).
3. The ectopic QRS complex is premature but has a normal shape unless there has been aberrant conduction.
4. They are generally followed by a compensatory pause; the SA node is not reset, and the next P wave occurs at its usual time.

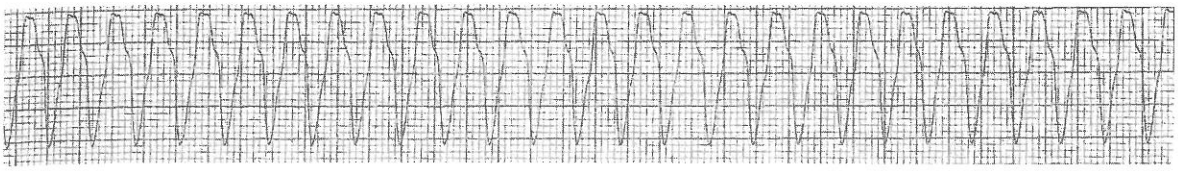
J. Premature ventricular contractions: appear as abnormal QRS complexes and T waves that occur in another underlying rhythm



Courtesy of Dr. Michael McCrea

1. Appear as abnormal QRS complexes and T waves that occur in another underlying rhythm
2. Characteristics
   a. Occur earlier than the next expected normal QRS
   b. Wider than a normal QRS (usually ≥0.12 second)
   c. The QRS morphology is generally bizarre.
   d. A preceding P wave is absent; however, retrograde conduction of a premature ventricular contraction can occasionally result in an inverted P wave after the QRS complex.
   e. The deflection of the ST segment and T wave is opposite that of the QRS.
   f. May occur in regular pattern, eg, bigeminy (as above)
   g. Generally followed by a compensatory pause; the sinoatrial node is not reset, and the next P wave occurs at its usual time.

K. Ventricular tachycardia: three or more consecutive premature ventricular contractions occurring at a rate >120 beats per minute



Courtesy of Dr. Michael McCrea

1. P waves are usually absent; when present, they are either retrogradely conducted or have no relationship to the QRS (AV dissociation).
2. QRS complexes are wide (≥0.12 second) and may be bizarre.
3. Fusion beats may be present; these are intermediate in appearance between a bizarre QRS complex and a normal QRS. When present, the diagnosis of ventricular tachycardia is certain.
4. Capture beats are rarely seen but, when present, confirm the diagnosis of ventricular tachycardia. Capture beats are the result of an atrial impulse penetrating the AV node from above to stimulate ("capture") the ventricles. Because ventricular conduction occurs over the normal pathways, the resulting QRS of the captured beat looks normal (narrow) in appearance.
5. Deflection of the ST segment and T wave is generally opposite that of the QRS complex.
6. Rate is >120 (usually 150–200) beats per minute.
7. Rhythm is generally regular, although beat-to-beat variation may occur.
8. QRS axis is generally constant.
9. Ventricular tachycardia is classified as "monomorphic" (QRS complexes look the same) or "polymorphic" (QRS complexes have varying morphology). Current therapeutic modalities are based on this classification (see Etiologies and Management of Dysrhythmias, pages 27–40).
10. Differentiation of supraventricular tachycardia with aberrancy from ventricular tachycardia
    a. P waves preceding QRS complexes favor aberrancy.
    b. A fully compensatory pause is more likely to occur with ventricular tachycardia.
    c. Response to vagal maneuvers (Valsalva maneuver, carotid sinus massage) may occur with aberrant supraventricular tachycardia, whereas ventricular tachycardia is unaffected. (Carotid sinus massage is contraindicated in older patients with a history of carotid disease/stroke or the presence of a carotid bruit.)
    d. Marked left axis deviation (>30°) suggests ventricular tachycardia; any QRS axis deviation >40° in either direction (or an upright QRS in aVR) favors ventricular tachycardia.
    e. QRS duration >0.14 second favors ventricular tachycardia.
    f. QRS concordance (all the QRS complexes from V_1 to V_6 are either positive or negative) strongly favors ventricular tachycardia.
    g. QRS morphology in lead V_1: an RS, R or qR with left "rabbit ear" taller than the right suggests ventricular tachycardia, whereas an RSR' pattern is more likely supraventricular tachycardia with aberrancy; negative QRS morphology in this lead with a wide R wave (>0.03 second), RS interval >0.07 second, and a slurred or notched S wave favors ventricular tachycardia.
    h. QRS morphology in lead V_6: R/S ratio <1, a qS or qR favors ventricular tachycardia.

i. Fusion and capture beats indicate AV dissociation and are practically diagnostic of ventricular tachycardia.

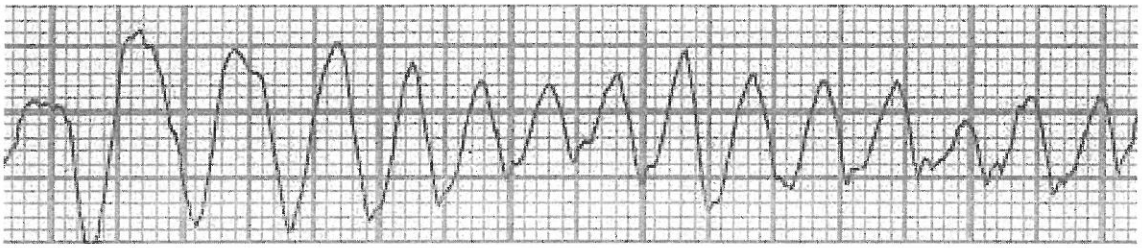
j. A bundle-branch pattern that varies suggests supraventricular tachycardia with aberrancy.

k. A history of prior heart disease (MI, CHF, coronary artery bypass graft) strongly favors ventricular tachycardia (likelihood 85%), as does a prior history of ventricular tachycardia.

l. Age ≥50 years old favors ventricular tachycardia, whereas age ≤35 years old favors an aberrant supraventricular tachycardia.

m. The vast majority of published criteria that can be used to diagnose ventricular tachycardia are fairly reliable; however, there are no reliable criteria to exclude ventricular tachycardia. Because the misdiagnosis of ventricular tachycardia can be deadly, if there is any doubt about the diagnosis whatsoever, always assume that a wide complex tachycardia is ventricular tachycardia and treat as such!

11. Torsades de pointes ("twisting of the points") is an atypical ventricular tachycardia in which the QRS axis swings from a positive to a negative direction in a single lead creating a "sine-wave" appearance. It originates from a single focus and is usually precipitated by drugs that prolong the QT interval, such as Class IA antidysrhythmics (procainamide, quinidine), Class IC antidysrhythmics (propafenone, fecainide), tricyclic antidepressants, droperidol, and the phenothiazines. The combined use of certain drugs such as terfenadine plus ketoconazole or erythromycin also prolong the QT interval and may therefore precipitate torsades. Other causes include hypomagnesemia and hypokalemia. The rate is typically 200–240 beats per minute.



Courtesy of Dr. Daniel Schwerin

L. Ventricular fibrillation



Courtesy of Dr. Michael McCrea

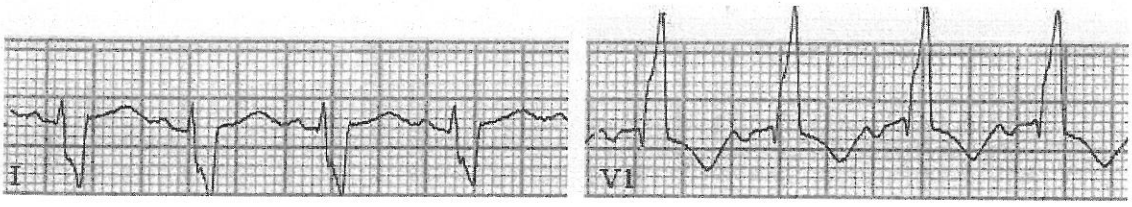
1. Most commonly recognized as a fine or coarse zigzag pattern without discernible P waves, QRS complexes, or T waves representing the presence of unorganized ventricular electrical activity
2. Sometimes the rhythm may look like ventricular tachycardia. The point is moot if the patient has no pulse and is unresponsive, because the treatment is the same (unsynchronized defibrillation).

M. Pulseless electrical activity (PEA)

1. Refers to a heterogeneous group of rhythms characterized by the presence of some type of electrical activity other than ventricular tachycardia or ventricular fibrillation in the absence of a perceptible pulse
2. Includes electromechanical dissociation, pseudoelectromechanical dissociation, idioventricular rhythms, ventricular escape rhythms, bradyasystolic rhythms, and postdefibrillation idioventricular rhythms
3. These dysrhythmias often occur in association with specific clinical conditions that if promptly identified and treated may result in a return of spontaneous circulation. Remember the "H's and T's"
   a. Hypovolemia
   b. Hydrogen ion, ie, acidosis
   c. Hypothermia
   d. Hypo/hyperkalemia
   e. Hypoxia
   f. Toxins
   g. Tamponade, cardiac
   h. Tension pneumothorax
   i. Thrombosis, coronary
   j. Thrombosis, pulmonary

N. Bundle-branch blocks (BBBs)

1. Abnormal conduction abnormalities (not rhythm disturbances) in which the ventricles depolarize in sequence (rather than simultaneously), thus producing a wide QRS complex (0.09–0.11 → incomplete BBB; ≥0.12 second → complete BBB) and an ST segment with a slope opposite that of the terminal half of the QRS complex
2. Right BBB is a unifascicular block in which ventricular activation is by way of the left bundle branch; the impulse travels down the left bundle, thus activating the septum from the left side (as it normally does in the absence of right BBB). This is followed by activation of the free wall of the left ventricle and, finally, the free wall of the right ventricle. Because of the two changes in direction, there is a tendency toward triphasic complexes in right BBB.



Courtesy of Dr. Michael McCrea

   a. Wide QRS complex (≥0.12 second)
   b. Triphasic QRS complex (rSR′ variant) in lead V_1
   c. Wide S waves in leads I, V_5, and V_6
   d. Normal septal Q waves in leads I and V_6 because the initial activation of the ventricle occurs in the normal manner
   e. T wave has a deflection opposite that of the terminal half of the QRS complex.
   f. Associated axis is variable; a normal axis, left axis deviation, or even right axis deviation may be present.

3. Left BBB is a bifascicular block in which the left ventricle is activated by way of the right bundle branch; the impulse travels down the right bundle, activating the septum and the free wall of the right ventricle, and then continues on in the same direction to activate the free wall of the left ventricle. Because the dominant forces are traveling in the same direction, there is a tendency toward monophasic QRS complexes.

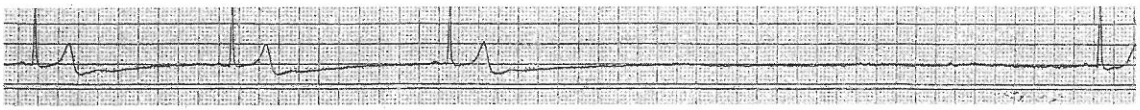


Courtesy of Dr. Michael McCrea

   a. Wide QRS complex (≥0.12 sec)
   b. Negative wave (QS or rS) in lead V_1
   c. Large, wide R waves in leads I, aVL, V_5, and V_6
   d. Absence of normal septal Q waves in leads I and V_6
   e. T wave has a deflection opposite that of the terminal half of the QRS complex.
   f. Associated left-axis deviation is most common and implies the presence of additional myocardial disease.

O. Sinoatrial block

1. Occurs when there is abnormal conduction between the sinus node and atrial muscle; recognized by the unexpected absence of a P wave and its associated QRS complex. Like AV block, sinoatrial block is also divided into first-, second-, and third-degree varieties.
2. First-degree sinoatrial block
   a. Impulse is delayed in its conduction from the sinoatrial node to the atria.
   b. Cannot be diagnosed from a surface 12-lead ECG
3. Second-degree sinoatrial block
   a. Some of the sinus node discharges are blocked.
   b. Recognized on an ECG as the absence of an expected P wave and its associated QRS complex
4. Third-degree sinoatrial block (sinus arrest)



Courtesy of Dr. Michael McCrea

   a. All of the sinus node discharges are blocked.
   b. On ECG, it may appear as a long sinus pause/arrest or a junctional/ventricular escape rhythm.

P. Sick sinus syndrome

1. An abnormality of cardiac impulse formation as well as intra-atrial and AV nodal conduction
2. Manifests as a wide variety of (or combinations of) bradyarrhythmias and tachyarrhythmias
3. Most common in the elderly

4. Presenting symptoms may include dizziness, palpitations, dyspnea, fatigue, lethargy, or syncope. Documentation of a bradyarrhythmia or tachyarrhythmia in association with these symptoms is the cornerstone of diagnosis.

Q. Atrioventricular blocks

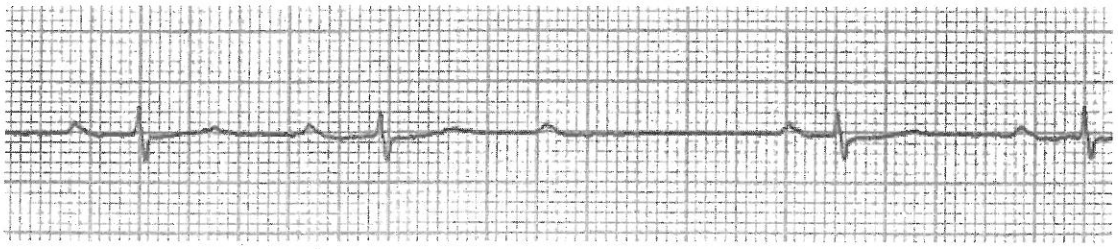
1. Occur when the conduction between the atria and ventricles is abnormal; the conduction delay can occur in the atria, the AV node, or the proximal His-Purkinje system.
2. First-degree AV block: normal AV conduction is slightly prolonged.

   Note PR 390 msec.



Courtesy of Dr. Michael McCrea

   a. P waves and QRS complexes are normal.
   b. There is a 1:1 relationship between the P and QRS.
   c. PR interval is prolonged (>0.20 second)
   d. The block is most often at the level of the AV node.
3. Second-degree AV block: some atrial impulses are not conducted.
   a. Mobitz Type I (Wenckebach)



      (1) P waves and QRS complexes are normal, but there are P waves without QRS complexes (nonconducted P waves).
      (2) PR interval progressively lengthens and the RR interval progressively shortens until a beat is dropped. This cycle repeats itself, producing a pattern referred to as "group beating."
      (3) The longest cycles (those of the dropped beats) are less than twice the length of the shortest cycles (those of the impulses following the dropped beats).
      (4) The block is almost always within the AV node.
   b. Mobitz Type II



Courtesy of Dr. Michael McCrea

(1) P waves are normal.

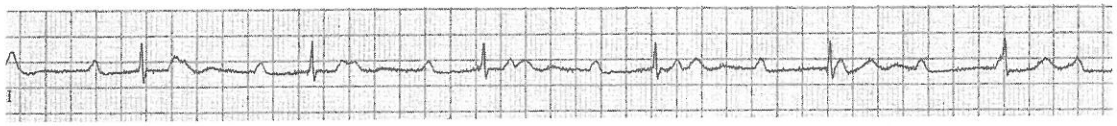
(2) QRS complexes are often (but not always) wide because of the common occurrence of a coexisting bundle-branch block.

(3) PR intervals (when they occur) are always the same duration.

(4) There are dropped beats.

(5) The block is below the level of the AV node, generally in the His-Purkinje system.

4. Third-degree AV block: no atrial impulses are conducted; the atria and ventricles beat independently of one another.



Courtesy of Dr. Michael McCrea

a. P waves appear normal.

b. Can occur at the level of the AV node, the bundle of His, or the bundle branches

c. QRS complexes may be narrow or wide, depending on the location of the block: if the block is located above the His bundle, the QRS complexes will be narrow and reflect a junctional escape rhythm, whereas if it is located at or below the bundle of His, the QRS complexes will be wide and reflect a ventricular escape rhythm.

d. There is no relationship between P waves and QRS complexes.

(1) There is an independent and regular atrial rate (constant PP interval) and a slower independent and constant ventricular rate (constant RR interval).

(2) The P waves are not related (not conducted) to the QRS complexes but rather "march through them" as if they were not there.

(3) The PR intervals are variable.

R. Preexcitation syndromes

1. Result from abnormal connections (accessory pathways) between the atria and ventricles. Impulses traveling down these pathways bypass all or part of the normal conduction system. This results in the ventricles being activated by atrial impulses sooner than would normally be anticipated (preexcitation) and is reflected by changes in the surface ECG. The ECG changes seen are determined by the exact pathway the impulse travels. Wolff-Parkinson-White syndrome and Lown-Ganong-Levine syndrome are the two major variants of preexcitation. Their characteristic ECG findings are listed below. Patients with preexcitation syndromes are prone to tachydysrhythmias (especially paroxysmal supraventricular tachycardia and atrial fibrillation) with very rapid ventricular rates (up to 300 beats per minute).

2. Wolff-Parkinson-White: the accessory pathway (bundle of Kent) connects the atria directly to the ventricles, completely bypassing the AV node and the infranodal conduction system.



Courtesy of Dr. Michael McCrea

a. Short PR interval (<0.12 second)
b. Delta wave (a slurred upstroke to the QRS)
c. Wide QRS
d. Secondary ST–T wave changes; deflection of the T wave may be opposite that of the QRS vector if the classic triad is present. (Otherwise, the QRS–T may appear normal.)

3. Lown-Ganong-Levine syndrome: the accessory pathway (James fibers) connects the atria directly to the proximal His bundle, completely bypassing the AV node.



Courtesy of Dr. Daniel Schwerin

a. Short PR interval (<0.12 second)
b. No delta wave
c. Normal QRS

III. ETIOLOGIES AND MANAGEMENT OF DYSRHYTHMIAS

A. Premature atrial contractions

1. There are multiple causes (drugs or underlying disease), but they may also occur as a normal variant.
2. Clinical significance
   a. Can precipitate supraventricular tachycardia, atrial fibrillation, and atrial flutter
   b. Most frequent cause of a pause on the ECG
3. In general, no treatment is indicated. If, however, the premature atrial contractions are frequent or symptomatic, treatment should be directed toward correcting the underlying cause.

B. Sinus tachycardia

1. There are multiple causes; common ones include:
   a. Anxiety (diagnosis of exclusion)
   b. Drugs (eg, cocaine)
   c. Fever
   d. Hypovolemia
   e. Hyperthyroidism
   f. Pulmonary embolism
   g. Anemia
   h. Hypoxia
   i. Pain
2. Management in most instances should be directed at finding and correcting the underlying cause. However, in the presence of an acute MI, patients with "inappropriate" tachycardia may benefit from the administration of ß-blockers to slow the heart rate. In the setting of cocaine toxicity, administration of a benzodiazepine may be helpful.

C. Sinus bradycardia

1. Common causes
   a. Acute inferior wall MI
   b. Vasovagal events (eg, vomiting)
   c. Drug effect (eg, ß-blockers, calcium channel blockers)
   d. Sick sinus syndrome
   e. A normal variant (especially in those individuals who exercise aerobically on a regular basis)
   f. Hypothermia
   g. Hypothyroidism
2. **Management**
   a. **Indicated for patients who demonstrate signs of hypoperfusion due to the bradycardia: those with shock, hypotension, ischemic chest pain, decreased mentation, or acute CHF**
   b. **Intervention sequence**
      (1) **Atropine 0.5–1 mg every 5 minutes as needed until a response is noted or a total of 0.03–0.04 mg/kg has been administered.**
         (a) **Should be used cautiously in patients with an acute MI, because it may worsen ischemia or precipitate ventricular tachycardia or ventricular fibrillation**
         (b) **Should also be used with caution in patients with Mobitz Type II second-degree AV block and new third-degree AV block with wide complexes → ↑ atrial rate → ↑ AV block → ↓ ventricular rate and ↓ blood pressure**
         (c) **Ineffective in patients with heart transplants; go directly to transcutaneous pacing and/or catecholamine infusion.**
         (d) **Can be parasympathomimetic in doses <0.5 mg, further decreasing heart rate**
      (2) **Transcutaneous pacing**
         (a) **The treatment of choice for patients who are unresponsive to atropine and for those with severe symptoms**
         (b) **Analgesics or sedatives may be required by some patients to be able to tolerate the pacing stimulus.**
      (3) **Dopamine 5–20 mcg/kg/min**
         (a) **Should be used when bradycardia is unresponsive to atropine and a transcutaneous pacer is not readily available**
         (b) **Also useful when associated hypotension is present**
      (4) **Epinephrine 2–10 mcg/min is the next agent of choice and is particularly useful in the presence of significant hypotension.**
      (5) **Transvenous pacing may be required if symptomatic bradycardia persists.**

D. Supraventricular tachycardia (SVT)

1. A generic term that refers to all tachydysrhythmias arising above the bifurcation of the bundle of His, including sinus tachycardia, atrial fibrillation, atrial flutter, multifocal atrial tachycardia, paroxysmal supraventricular tachycardia, and nonparoxysmal junctional tachycardia. It arises from reentry or an ectopic pacemaker in the atria. Most clinicians, however, use the term SVT to refer to AV nodal reentry tachycardia and other undetermined supraventricular rhythms. In the discussion that follows, SVT is used in this sense of the word. Management of other specific forms of SVT (such as atrial fibrillation) is discussed separately.

2. Causes
   a. Preexcitation syndromes (Wolff-Parkinson-White and Lown-Ganong-Levine)
   b. Mitral disease (prolapse, stenosis)
   c. Digitalis toxicity
   d. Drugs (eg, alcohol, tobacco, caffeine)
   e. Acute MI and pericarditis
   f. Hyperthyroidism
   g. Rheumatic heart disease
3. **Management is determined primarily by the patient's hemodynamic stability and secondarily by the width of the QRS complex.**
   a. **Hemodynamically compromised patients (those with hypotension, ischemic chest pain, a decrease in mental status, or acute CHF) with a narrow complex SVT should be sedated (if possible) and treated with synchronized cardioversion. Start with 50 joules. (In the toxic digitalis patient, cardioversion is potentially hazardous and should be avoided if at all possible. Use it only in situations of last resort.**
   b. **Vagal maneuvers and pharmacologic therapy may be used in the hemodynamically stable patient with narrow complex SVT.**
      (1) **Vagal maneuvers (such as carotid sinus massage [should not be done if digitalis toxicity has not been excluded] or Valsalva maneuver) increase vagal tone and may be effective in either terminating the dysrhythmia or slowing the ventricular rate enough to uncover the actual underlying rhythm. These maneuvers should be attempted before starting pharmacologic therapy and may also be used to supplement it. The vagal maneuver of choice, ie, the most effective, is the Valsalva maneuver.**
      (2) **Adenosine, because of its safety profile, is the drug of choice for the hemodynamically stable patient with narrow complex SVT. It is an ultra-short-acting AV nodal blocker that is very effective in converting SVT. Its major advantages over verapamil are its short half-life (<10 seconds) and its lack of hypotensive and myocardial depressant effects. Although it does produce adverse effects (flushing, dyspnea, chest pain), they are transient. Recurrence of SVT, however, is common (up to 50%–60% of patients). Adenosine does have several significant drug interactions. Its effects are antagonized by the methylxanthines (theophylline, caffeine) and potentiated by dipyridamole and carbamazepine. Therefore, large doses of adenosine may be required in the presence of methylxanthines, whereas smaller doses (or an alternative agent) should be used in the presence of dipyridamole and carbamazepine.**
      (3) **Calcium channel blockers (eg, diltiazem, verapamil) are as effective as adenosine but slower in onset and produce more significant adverse effects (decreased myocardial contractility and peripheral vasodilation). Calcium channel blockers should not be used concomitantly with IV ß-blockers and are contraindicated in patients with wide complex tachycardias, atrial fibrillation with Wolff-Parkinson-White, sick sinus syndrome, and advanced AV block.**
         (a) **Pretreatment with a fluid bolus and calcium chloride (0.5–1 g IV over several minutes) are useful in preventing the hypotension induced by the vasodilatory effects of verapamil.**
         (b) **Diltiazem seems to be as effective as verapamil in the treatment of narrow complex SVT and has the advantage of producing less myocardial depression.**

(4) ß-blockers such as esmolol, metoprolol, or propranolol are also effective in the treatment of narrow complex SVT. Esmolol has the advantage of being cardioselective as well as having a very short half-life. Propranolol is the drug of choice for SVT secondary to thyrotoxicosis, because it partially blocks the conversion of T_4 and T_3. Avoid these drugs in patients with COPD, asthma, or CHF; in those who have received IV calcium channel blockers; and in those with atrial fibrillation with Wolff-Parkinson-White, sick sinus syndrome, and advanced AV block.

(5) Digoxin is vagotonic. Compared with the other agents listed above, its effects are mild and have a much slower onset (may take several hours or more to work). Digoxin should be avoided if cardioversion is being considered.

(6) Magnesium sulfate, phenytoin, and lidocaine are the drugs of choice for ectopic SVT caused by digitalis toxicity. Management should also include correction of hypokalemia (if present) and discontinuation of digitalis. In the presence of hemodynamic instability (or potentially lethal digitalis intoxication), administration of Fab fragments should be considered.

(7) Other antidysrhythmic agents (eg, procainamide, amiodarone, sotalol) may also be effective.

(8) Patients who do not respond to drug therapy may be treated with synchronized cardioversion (as described above) or overdrive cardiac pacing.

c. Drug dosages and administration

(1) Adenosine 6 mg rapid IV push in a proximal vein followed by a 20-mL bolus of normal saline; if there is no response after 1–2 minutes, double the dose to 12 mg.

(2) Verapamil 2.5–5 mg IV over 2–3 minutes; a second dose of 5–10 mg may be given in 15–30 minutes if necessary.

(3) Diltiazem 0.25 mg/kg IV over 2 minutes, followed in 15 minutes by a second bolus of 0.35 mg/kg if the first bolus was tolerated but ineffective. Smaller dosages should be considered in elderly patients.

(4) Esmolol 300–500 mcg/kg bolus over 1 minute followed by an infusion of 50 mcg/kg/min; the loading dose may need to be repeated and the infusion rate increased by 50 mcg/kg/min every 5 minutes as needed to a maximum of 200 mcg/kg/min.

(5) Metoprolol 5 mg IV over 2 minutes; may be repeated twice every 5 minutes for total of three doses.

(6) Propranolol 1 mg IV over 1 minute; this dose may be repeated every 5 minutes up to a total dosage of 0.1–0.5 mg/kg.

(7) Digoxin 0.5 mg IV push initially, with repeated doses of 0.25 mg every 30–60 minutes as needed; total dosage should not exceed 0.02 mg/kg.

(8) Magnesium sulfate 1–2 g slow IV push over 1–2 minutes followed by an infusion of 1–2 g/hr

(9) Phenytoin 18 mg/kg IV bolus infusion; dissolve dosage in normal saline and administer at a rate of 50 mg/min or less.

(10) Lidocaine 1–1.5 mg/kg bolus infusion, repeat dosages 0.5–0.75 mg/kg every 5–10 minutes, to maximum bolus dose of 3 mg/kg. This can be followed by maintenance infusion of 1–4 mg/min.

d. Patients with wide complex tachycardia should be presumed to have ventricular tachycardia.

(1) If unstable → synchronized cardioversion

(2) **If stable → procainamide or amiodarone**

(a) **Both convert SVT or ventricular tachycardia.**

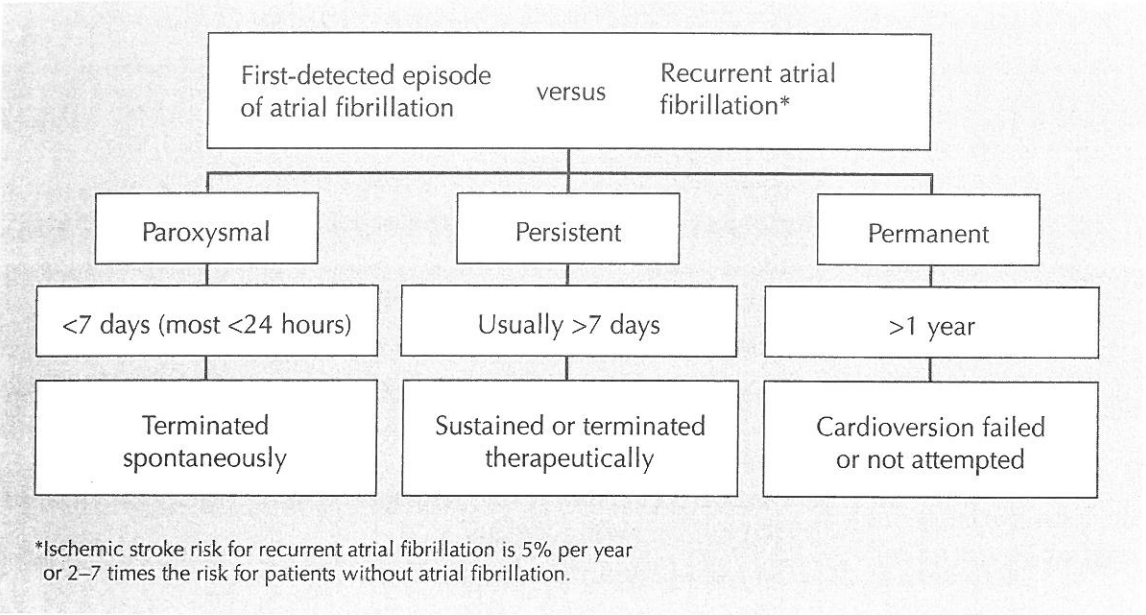
(b) **Both should be avoided in patients with tricyclic antidepressant overdose or in the setting of toxicity from other sodium-channel blockers.**

(c) **Adenosine may initially slow either rhythm, but dysrhythmia may recur (short therapeutic effect of the drug).**

(d) **If drug therapy fails → synchronized cardioversion**

E. Atrial fibrillation (most common SVT)

1. Identify the type of atrial fibrillation the patient has by determining the probable duration of the dysrhythmia.



2. **Search for reversible causes and treat any underlying medical condition; then determine the risk of subsequent stroke. Conditions with high risk of cardiogenic thromboembolism:**
   a. **Cardiac surgery**
   b. **Acute MI**
   c. **Hyperthyroidism**
   d. **Myocarditis**
   e. **Acute pulmonary disease**
3. **Plan the treatment using the following criteria:**
   a. **Cardiovascular stability**
   b. **Duration of the dysrhythmia**
   c. **Underlying cause/condition**
   d. **Presence/absence of an accessory pathway**
4. **There are fundamentally two ways to manage atrial fibrillation: restore and maintain sinus rhythm, or allow atrial fibrillation to continue and ensure that the ventricular rate is controlled.**
   a. **Unstable patients → immediate electrical synchronized cardioversion**
   b. **Stable patients with significant symptoms → pharmacologic therapy**

(1) Control the ventricular rate first (choose one)
  (a) Calcium channel blocker (eg, verapamil, diltiazem)
  (b) ß-blocker (eg, esmolol, atenolol, metoprolol)
  (c) Digoxin
  (d) Amiodarone
  (e) The presence of Wolff-Parkinson-White syndrome is a special circumstance requiring changes in treatment protocols (see page 40).

(2) Cardiovert the dysrhythmia based on the duration of atrial fibrillation.
  (a) <48 hours duration
    i. Normal cardiac function: perform electrical cardioversion or use one of the following agents:
      • Amiodarone
      • Ibutilide
      • Flecainide
      • Propafenone
      • Procainamide
    ii. Compromised cardiac function: perform DC cardioversion or use amiodarone
  (b) >48 hours duration (higher risk of systemic embolization)
    i. Avoid immediate cardioversion if possible.
    ii. If early cardioversion (within 24 hours) is anticipated, consider starting heparin and consulting cardiology for transesophageal echocardiography to exclude an atrial clot.
    iii. If delayed cardioversion is the best option, anticoagulation for 3 weeks is indicated before cardioversion.

5. Discussion of selected treatment options
  a. Hemodynamically compromised patients should be sedated (if possible) and treated with synchronized cardioversion; start with 120 joules. In patients who have digitalis toxicity or are hypokalemic, cardioversion is potentially hazardous; use only as a last resort and start with lower joules. If the patient has hypertrophic cardiomyopathy (or has been in atrial fibrillation >48 hours), immediate systemic heparinization is also indicated.
  b. Hemodynamically stable patients with atrial fibrillation of acute (<48 hours) onset (paroxysmal or persistent atrial fibrillation) should be managed with pharmacologic therapy, the primary goal of which is to slow the ventricular response rate to 100 beats per minute or less.
    (1) First-line agents include calcium channel blockers (eg, diltiazem, verapamil) and ß-blockers (eg, esmolol, metoprolol).
    (2) Digoxin may also be used, but it is much slower in onset and is not likely to be effective if the sympathetic tone is high; it should be considered a second-line agent.
    (3) Magnesium is also effective in slowing the ventricular rate and may be used in combination with one of the above agents as adjunct therapy.
    (4) Once the ventricular rate is controlled, a pharmacologic agent can be given to chemically convert the patient to sinus rhythm. Ibutilide is effective but associated with a significant risk of inducing torsades de pointes. Procainamide is also effective at pharmacologic cardioversion. (Early pharmacologic and electrical cardioversion patients with acute-onset atrial fibrillation is gaining acceptance in emergency medicine.)

c. **Patients with atrial fibrillation >2 days duration should, when stable, receive anticoagulation before pharmacologic or electric cardioversion is attempted to decrease the risk of arterial embolization of an intra-atrial thrombus.**

F. Atrial flutter

1. Causes are essentially the same as for atrial fibrillation, but it is most often associated with postcardiac surgery and peri-infarction periods.
2. Usually a transitional rhythm between sinus rhythm and atrial fibrillation
3. Management is determined by the patient's cardiovascular stability, duration of the dysrhythmia and whether or not the patient has an accessory pathway.
   a. Hemodynamically unstable patients should be sedated (if time permits) and treated with synchronized cardioversion; start with 50 joules.
   b. Hemodynamically stable patients should be managed with Vagal maneuvers and pharmacologic therapy.
      (1) Vagal maneuvers or adenosine may be of diagnostic value; by inducing a transient AV nodal blockade, they may reveal the characteristic flutter waves of this rhythm and confirm the diagnosis.
      (2) Once the diagnosis is confirmed, rate control should be accomplished with a calcium channel blocker (eg, diltiazem, verapamil) or a □-blocker (eg, esmolol, metoprolol). Digoxin may also be used but is considered a second-line drug. Magnesium may be used in combination with one of the above agents as adjunctive therapy.
      (3) Once the ventricular response rate is controlled, pharmacologic conversion to sinus rhythm can be achieved with procainamide or quinidine.

G. Multifocal atrial tachycardia

1. Causes
   a. Decompensated COPD (most common)
   b. CHF
   c. Sepsis
   d. Theophylline toxicity
   e. Asthma
2. Management
   a. Primarily aimed at correcting the underlying disease process
      (1) Correct hypoxia with supplemental oxygen (and bronchodilator therapy) in a patient with COPD, and
      (2) Evaluate for theophylline toxicity.
   b. If the above measures are unsuccessful and the patient is symptomatic, other modalities that may be used:
      (1) Calcium channel blockers (eg, diltiazem, verapamil) are usually effective in slowing the ventricular rate and decreasing atrial ectopy, and may produce conversion to a sinus rhythm in some patients.
      (2) Magnesium sulfate decreases atrial ectopy.
      (3) ß-blockers are generally not recommended, because they may worsen the underlying pulmonary disease process.
   c. Digoxin and cardioversion are usually ineffective.

H. Junctional premature contractions

1. Primary causes
   a. Digitalis toxicity
   b. Coronary artery disease
   c. CHF
   d. Acute MIs (especially inferior wall MIs)
2. Treat the underlying cause; if junctional premature contractions precipitate more lethal dysrhythmias, consider using IV procainamide.

I. Premature ventricular contractions (PVCs)

1. Causes
   a. Hypokalemia
   b. Hypomagnesemia
   c. Hypoxia
   d. MI
   e. Drugs
      (1) Alcohol, tobacco, caffeine
      (2) Cocaine
      (3) Digitalis or quinidine toxicity (PVCs are most common dysrhythmia with digitalis toxicity)
      (4) Methylxanthines (commonly used by patients with asthma or COPD)
   f. Hyperthyroidism
   g. CHF
   h. Cardiomyopathy
   i. Mechanical: PVCs are not uncommon when a catheter is placed in the right ventricle.
   j. Myocardial contusion
2. Management is dictated by the underlying cause and is not indicated in all cases.
   a. Patients who are asymptomatic and have PVCs of unknown cause should not be treated (particularly when the PVCs are an incidental finding); PVCs in this setting may represent a normal variant. Isolated PVCs occur in as many as 50% of young healthy patients and increase in frequency with age.
   b. When an underlying cause is identified (hypokalemia, hypomagnesemia, hypoxia, etc), therapy should be directed toward correcting the underlying problem (rather than suppressing PVCs). This is usually sufficient.
   c. Mechanical causes, if present, should also be corrected.
      (1) A central line catheter that is located in the right ventricle may induce PVCs; withdraw the catheter, or advance it out of the ventricle.
      (2) To reduce the likelihood of inducing PVCs during Swan-Ganz placement, inflate the balloon to cover the catheter tip while advancing into the right ventricle; then deflate the balloon to avoid floating into the ventricular outflow tract.
      (3) If these measures fail to ameliorate the PVCs and the patient is symptomatic, lidocaine may be administered.
   d. "Escape" PVCs (those associated with bradycardia) should be treated with atropine (not lidocaine), because administration of lidocaine under these circumstances may suppress the existing functioning rhythm.

e. Management of PVCs occurring in association with an acute MI or ischemia is more controversial. Optimal treatment of the underlying ischemia/infarction with oxygen, nitroglycerin, morphine, aspirin, and fibrinolytic therapy is clearly the first priority. If these measures fail, most authors currently recommend a conservative course of watchful waiting.

f. In general, the identification of PVCs should prompt a search for and treatment of the underlying cause, rather than simply pharmacologic suppression of the PVCs. Empiric use of magnesium is sometimes recommended. If short bursts of frequent PVCs are occurring, consider empiric administration of □-blockers (to decrease adrenergic stimulation that often causes ventricular dysrhythmias).

g. Pharmacologic agents

(1) Lidocaine has historically been used as a first-line agent, though it is rarely used empirically now. If the decision is made to use lidocaine, administer a bolus of 1–1.5 mg/kg followed by a 2–4 mg/min drip. Repeat boluses of 0.5–0.75 mg/kg may be given every 5–10 minutes as needed to a maximum total dosage of 3 mg/kg.

(2) Procainamide may be tried if lidocaine is ineffective or contraindicated. It is administered at an infusion rate of 20–30 mg/min until a favorable response is noted, the QRS widens by 50% of its original width, hypotension develops, or a total dosage of 17 mg/kg has been administered. This should be followed by a maintenance drip of 1–4 mg/min.

(3) Magnesium sulfate is often effective in decreasing the frequency of PVCs. The dose is 1–2 g slow IV push over 1–2 minutes followed by an infusion of 1–2 g/hr.

J. Ventricular tachycardia

1. The causes are basically the same as those for PVCs. The most common causes are ischemic heart disease and acute MI.

2. Management depends on the status of the patient and QRS morphology.

a. If there is no pulse for either monomorphic or polymorphic ventricular tachycardia, treat like ventricular fibrillation: begin CPR and defibrillate as soon as a defibrillator is available. Start with 200 joules.

b. If the patient has a pulse but is hemodynamically unstable (hypotensive, decreased mental status, ischemic chest pain, acute CHF), synchronized cardioversion is indicated. Sedate first if time allows. Start with 100 joules, and increase if unsuccessful.

c. If the patient is clinically stable (awake, pain free, and has good blood pressure), pharmacologic therapy is appropriate. Patients who do not convert with these agents should be sedated and cardioverted. Start with 100 joules. Assuming the patient is stable enough for medications, the following are recommended:

(1) Monomorphic ventricular tachycardia: procainamide, amiodarone, or sotalol

(2) Polymorphic ventricular tachycardia

(a) If the baseline QT interval is normal, all of the above agents are considered reasonable.

(b) If the QT interval is prolonged, it should be assumed that the dysrhythmia is torsades de pointes (atypical ventricular tachycardia). Class 1A and 1C antidysrhythmic agents are contraindicated; they prolong ventricular repolarization, which can worsen torsades.

i. The therapy of choice is magnesium sulfate 1–2 g IV over 1–2 minutes followed by an infusion of 1–2 g/hr; it is nondysrhythmogenic and rapidly effective.

ii. Overdrive electrical pacing (transcutaneous or transvenous) is also effective by increasing the heart rate, thereby shortening ventricular repolarization.

iii. Sustained torsades or unstable patients should be treated with direct cardioversion, ie, not synchronized; start with 200 joules, and begin a magnesium infusion after cardioversion.

(3) Note: If a patient with polymorphic ventricular tachycardia or torsades is unstable but has a pulse, cardioversion is indicated. Because of concerns in delay of cardioversion due to trouble with synchronization, even in a patient with a pulse, use direct cardioversion, ie, unsynchronized and defibrillation doses starting at 200 joules. Once cardioversion is achieved, antidysrhythmic therapy with, amiodarone or procainamide should be given. If the patient had torsades, magnesium should be used instead, as noted above.

d. For patients who have cardioverted back to sinus rhythm or who are experiencing intermittent ventricular tachydysrhythmias, the use of ß-blockers should also be considered. ß-blockers decrease the adrenergic tone that contributes to ventricular tachydysrhythmias.

3. It is a common misconception that all patients with ventricular tachycardia will appear clinically unstable; stable patients with a wide complex tachycardia are frequently assumed to have SVT with aberrancy rather than ventricular tachycardia. This is an inaccurate assumption.

a. Ventricular tachycardia cannot be differentiated from SVT with aberrancy based on clinical symptoms and vital signs.

b. Unstable patients with either rhythm should be cardioverted; it is effective in both cases.

c. An ECG should be obtained in all stable patients.

(1) Examine for evidence that favors one dysrhythmia over the other and treat accordingly.

(2) If unable to decide, always assume it is ventricular tachycardia and treat as above based on QRS morphology and cardiac function.

d. Adenosine will convert some forms of ventricular tachycardia to a sinus rhythm; therefore, use of adenosine as a diagnostic measure to distinguish ventricular tachycardia from SVT with aberrancy is not recommended.

K. Ventricular fibrillation (and pulseless ventricular tachycardia)

1. Causes

Table 3: The Five H's and the Five T's

Hypovolemia (most common)	Tablets (drug overdose)
Hypoxemia	Tamponade (cardiac)
Hydrogen ion (acidosis)	Tension pneumothorax
Hyper- and hypokalemia	Thrombosis, coronary (acute coronary syndrome)
Hypothermia	Thrombosis, pulmonary embolism

2. **Management**

a. Initial therapy is *immediate defibrillation*. Time is of the essence. Chest compressions should be performed while setting up for defibrillation. Begin shocks at maximum joules (360 joules if using a monophasic defibrillator, 200 joules if using a biphasic defibrillator). Escalating dosages of shocks and stacked shocks are no longer

indicated. After the defibrillation, immediately begin compressions at 100 times per minute for 2 minutes without checking for pulse or checking monitor. For persistent ventricular fibrillation/pulseless ventricular tachycardia, continue to follow the pattern of defibrillation → compressions for 2 minutes → check pulse and monitor. A key point is to minimize interruptions in compressions.

b. **If defibrillation is unsuccessful, follow the "CABs" as summarized in the ACLS 2010 Guidelines.**

(1) **Circulation → high quality chest compressions with minimal interruptions for pulse checks, advanced airway insertion, or other procedures**

(2) **Airway → ensure open airway, place an advanced airway if unable to adequately bag-valve-mask; 30 chest compressions to 2 breaths for adults regardless of one or two rescuers. Performance of endotracheal intubation or bag-valve-mask ventilation should not interrupt chest compressions.**

(3) **Breathing → if an advanced airway is placed, confirm endotracheal tube placement, secure the tube, and confirm oxygenation and ventilation using monitored end-tidal CO_2 capnography.**

c. Although no reduction in mortality has been shown with use of any IV medications in cardiac arrest, epinephrine is still recommended every 3–5 minutes as needed in doses of 1 mg for patients who remain pulseless. At this point, an active search for one of the reversible causes listed above should be sought and, if found, treated. Vasopressin may be used instead of the first dose of epinephrine at a one-time dose of 40 units IV.

d. If ventricular fibrillation or pulseless ventricular tachycardia persists despite defibrillation, epinephrine, chest compressions, and treatment of any obvious reversible cause (eg, hyperkalemia), an antidysrhythmic agent can be administered. However, no antidysrhythmics have been demonstrated to improve survival. Lidocaine was removed from the 2010 ACLS pulseless arrest algorithm, leaving amiodarone as the only recommended agent for refractory ventricular fibrillation/pulseless ventricular tachycardia. Lidocaine may be given if amiodarone is not available. Exception: If the patient is being treated for pulseless torsades de pointes, magnesium is the drug of choice.

e. Dosages

(1) **Amiodarone 300 mg IV push; may repeat once at 150 mg IV push followed by a 1 mg/min infusion for 6 hours, then 0.5 mg/min for 18 hours as needed**

(2) **Magnesium sulfate 1–2 g IV push for torsades de pointes, known or suspected hypomagnesemia, or severe refractory ventricular fibrillation**

L. Pulseless electrical activity

1. Refers to a heterogeneous group of rhythms characterized by the presence of some sort of electrical activity (other than ventricular tachycardia or ventricular fibrillation) in the absence of a palpable pulse
2. Causes: same as those for ventricular fibrillation/pulseless ventricular tachycardia
3. Management
   a. Begin resuscitation: perform CPR, intubate, start an IV line.
   b. Search for and treat the underlying cause when possible. The approach should be guided by the clinical findings, medical history, and circumstances that preceded the development of the rhythm. Appropriate measures include:
      (1) Ventilate with 100% oxygen to treat hypoxia.
      (2) Listen for breath sounds in both lung fields, and assess the ease of manual ventilation to exclude tension pneumothorax.

(3) Administer a fluid bolus to correct hypovolemia.
(4) Assess the ECG for evidence of an acute MI and hyperkalemia.
(5) Check core body temperature to exclude hypothermia.
(6) Look for distended neck veins to exclude tension pneumothorax and cardiac tamponade.
(7) Perform bedside ultrasound if available to exclude cardiac tamponade.
(8) Obtain brief history from EMS or the patient's family or friends regarding the possibility of drug overdose.

c. Administer epinephrine 1 mg every 3–5 minutes. Atropine was removed from the 2010 ACLS pulseless electrical activity algorithm because of absence of proven reduction in mortality.

M. Sick sinus syndrome

1. Causes
   a. Ischemia
   b. Cardiomyopathy
   c. Myocarditis
   d. Trauma
   e. Atherosclerosis
   f. Aging
2. Management depends on the hemodynamic stability of the patient. If symptoms of hypoperfusion are present, rate stimulation with atropine, a pacemaker (for bradydysrhythmias), or rate control with a calcium channel blocker, ß-blocker, or digoxin (for tachydysrhythmias) can be cautiously attempted (excessive tachycardia or bradycardia may result). Hemodynamically stable patients should be referred to a cardiologist for demand pacemaker insertion and antidysrhythmic therapy.

N. Atrioventricular blocks

1. Causes of impaired conduction
   a. Acute MI (inferior wall or anterior wall)
   b. Drugs: digitalis, lidocaine, phenytoin, procainamide, quinidine, ß-blockers, calcium channel blockers, magnesium infusion (iatrogenic)
   c. Inflammation: myocarditis, endocarditis
   d. Hyperkalemia
   e Hypermagnesemia
   f. Hypothermia
   g. Congenital conduction defects
2. Management
   a. First-degree AV block is often a normal variant. Other causes include increased vagal tone, digitalis toxicity, and myocarditis; specific therapy is not required.
   b. Second-degree AV block
      (1) Mobitz Type I (Wenckebach)
         (a) Usually transient
         (b) Often seen in the setting of an acute inferior wall MI but can also be caused by digitalis toxicity, increased vagal tone, and myocarditis

(c) **Unless the patient is symptomatic, specific therapy is generally not required.**

(d) **Symptomatic patients should be treated with atropine; if this fails, transcutaneous pacing is indicated.**

(2) **Mobitz Type II**

(a) **A more severe form of block; implies an organic lesion in the infranodal conduction system and is often permanent**

(b) **Usually occurs in association with an acute anteroseptal MI and can abruptly progress to complete block**

(c) **These patients require pacing; transcutaneous pacing may be used as a bridging device, while preparations are made for insertion of a transvenous pacemaker. Atropine should be avoided (it can accelerate the atrial rate and produce a relative increase in the AV block → ↓ ventricular response rate and ↓ blood pressure).**

c. **Third-degree (complete) AV block**

(1) **Nodal (narrow complex) third-degree AV block occurs in association with an acute inferior wall MI or drug toxicity and is usually transient. It should be treated like second-degree Mobitz Type I AV block with atropine or transcutaneous pacing.**

(2) **Infranodal (wide complex) third-degree AV block often occurs in association with an acute anterior wall MI and implies significant structural damage to the infranodal conduction system. It almost always requires the insertion of a transvenous pacemaker. Transcutaneous pacing or catecholamines (dopamine or epinephrine infusions) may be used as a bridging device while awaiting pacemaker insertion. Atropine should be used cautiously (if at all) in this setting and is probably best avoided.**

O. Wolff-Parkinson-White (WPW) syndrome

1. Recognition
   a. PR interval <0.12 seconds
   b. Delta wave: a slurred initial upstroke of the QRS complex
   c. Wide and/or bizarre QRS complex (≥0.10 seconds)
   d. An episodic tachycardia >200 beats per minute in an adult is very suspicious of an accessory pathway syndrome.
   e. Patients with WPW frequently do not have all of the classic features described above on their surface ECG, particularly if they are in sinus rhythm at the time of evaluation and conduction is occurring through the AV node in the normal fashion (concealed tract).
2. The primary significance of WPW syndrome is that it predisposes to development of tachydysrhythmias (particularly atrial fibrillation).
   a. Patients with WPW have two parallel conducting pathways with different refractory periods, the AV node and an accessory pathway called the bundle of Kent. This predisposes them to the development of circus-movement tachycardias, in which an impulse is conducted anterograde to the ventricles by one pathway and retrograde back up to the atria by the other.
   b. When conduction is occurring anterograde down the AV node and retrograde back up the accessory pathway, ie, orthodromic tachycardia, the QRS complex *appears normal.* However, when the impulse is being conducted anterograde through the accessory pathway and retrograde through the AV node, the QRS complex is wide and called antidromic tachycardia.

c. In the presence of antidromic conduction, the normal restraining effect of the AV node on conduction is lost, and rapid ventricular response rates (>200 beats per minute) can occur. This is particularly dangerous in atrial fibrillation, in which ventricular response rates can exceed 300 beats per minute and degeneration into refractory ventricular fibrillation can occur.

3. **Management of supraventricular tachydysrhythmias in patients with WPW is determined by the patient's cardiovascular stability, specific presenting dysrhythmia, the width of the QRS complex, and (with atrial fibrillation) how long the patient has been in it.**
   a. **WPW with a narrow complex SVT (orthodromic tachycardia; most common presentation) is treated in the same manner as other re-entrant SVTs.**
      (1) **Treat hemodynamically compromised patients with synchronized cardioversion. Start with 50 joules, and pretreat with a sedating agent.**
      (2) **In hemodynamically stable patients, vagal maneuvers and drugs that slow conduction through the AV node (adenosine, calcium channel blockers or ß-blockers, procainamide, or amiodarone) should be used. Adenosine is the most effective agent given its rapid onset and strong AV nodal–blocking effects. IV procainamide is also effective but is slower in onset.**
   b. **WPW with a wide complex SVT (antidromic tachycardia)**
      (1) **If the patient is hemodynamically compromised, treat with cardioversion.**
      (2) **In stable patients, IV procainamide or amiodarone can be used effectively. Because the rhythm will look just like ventricular tachycardia (regular wide complex tachycardia), lidocaine may be chosen as first-line treatment, and it is unlikely to have any effect on the rhythm or hemodynamic status.**
      (3) **AV nodal–blocking agents (especially calcium channel blockers and ß-blockers, but also digitalis and adenosine) are contraindicated and should be avoided.**
   c. **WPW with atrial fibrillation or flutter (regardless of QRS duration)**
      (1) **Atrial fibrillation with WPW is characterized by an extremely rapid (rates typically 150–300 beats per minute), irregularly irregular tachycardia with QRS complexes that randomly vary in width and amplitudes, distinguishing this rhythm from ventricular tachycardia and from atrial fibrillation with bundle branch block. Atrial flutter with WPW is characterized by extremely rapid ventricular rates (often >250 beats per minute) with wide or narrow QRS complexes. The rapidity of the rate distinguishes this rhythm from typical ventricular tachycardia and from typical atrial flutter.**
      (2) **In hemodynamically compromised patients, cardioversion is the treatment of choice.**
      (3) **In hemodynamically stable patients, IV procainamide is the drug of choice, because it suppresses conduction through the accessory pathway.**
      (4) **All AV nodal–blocking agents should be avoided, because they can accelerate conduction through the accessory pathway and increase heart rate. These agents include adenosine, calcium channel blockers, ß-blockers, and digoxin. The use of amiodarone in these patients is controversial. Amiodarone does have both ß-blocking and calcium channel–blocking effects, which could potentially block the AV node and worsen the dysrhythmia.**

IV. PACEMAKERS

A. Emergency pacing techniques

1. Emergency cardiac pacing can be accomplished via transcutaneous, transesophageal, or transvenous electrodes.
2. Transcutaneous pacing is the technique of choice in the emergency care setting, particularly in the presence of an acute MI in patients who have received (or may receive) thrombolytic therapy. It is the most easily and rapidly applied technique as well as the least invasive.

B. Indications for temporary cardiac pacing

1. Emergent
   a. Hemodynamically unstable bradycardia
   b. Bradycardia associated with malignant escape rhythm that does not respond to pharmacologic therapy
   c. Overdrive pacing of a refractory tachydysrhythmia
   d. The pacing of asystole is no longer recommended.
2. Prophylactic (standby kit at bedside)
   a. Stable bradycardias (asymptomatic or those that responded to initial drug therapy)
   b. The presence of one of the following in the setting of an acute MI
      (1) Symptomatic sinus node dysfunction
      (2) Mobitz Type II second-degree AV block
      (3) Complete heart block
      (4) Newly acquired or age-indeterminate left bundle-branch block, right bundle-branch block, alternating bundle-branch block, or bifascicular block

C. Tips for temporary transvenous pacemaker placement

1. The ideal location of the catheter tip is lodged in the trabeculae of the apex of the right ventricle.
2. The right internal jugular is the preferred access site, because it provides the most direct route to the right ventricle.
3. Although more time consuming and not always possible, insertion using fluoroscopic or ECG guidance is preferred to blind placement (which is less reliable in achieving proper catheter positioning).
4. When ECG guidance is used, the V lead is connected to the distal lead of the pacing catheter with a standard connector or alligator clip. Then, while monitoring the V lead, the pacing catheter is advanced and the size of the P wave and QRS complex is observed to determine location of the catheter tip; the size of these wave forms will increase when the corresponding heart chambers are entered. The development of ST elevation ("current of injury") indicates successful placement of the catheter tip.
5. Pacing results in an abnormal QRS morphology. When the catheter tip is properly placed in the right ventricle, this ventricle is stimulated first, while stimulation of the left ventricle is delayed. This produces a left bundle-branch block pattern on the surface ECG.
6. After pacemaker placement, chest radiographs (AP and lateral) should be obtained to confirm appropriate placement and exclude a procedural pneumothorax.

D. Pacemaker failure

1. Tools for evaluating pacemaker malfunction
   a. ECG to confirm that appropriate sensing and capture are occurring and to indirectly assess the position of the pacing catheter by evaluating QRS morphology
   b. Chest radiograph (PA and lateral) to evaluate lead position, exclude cardiac perforation, and look for lead fracture (not always visible)
   c. Pacemaker magnet: application of the magnet over the pacemaker turns off the sensing function (via the Reed switch) and temporarily converts the pacemaker from the demand (synchronous) mode to the fixed-rate (asynchronous) mode → continuous asynchronous pacing at a specified rate (the magnet rate), which is usually around 70. This allows assessment of whether the pacing function is intact and whether the pacing stimulus can capture the myocardium. This maneuver is especially helpful in cases in which the baseline ECG does not reveal any pacemaker spikes. Moreover, it also allows assessment of the battery status; a decrease in the magnet rate suggests battery depletion.
2. Signs of pacemaker failure
   a. Slowing of the pacing rate is due to battery depletion. Replacement is urgent if the rate is 10% below the set point.
   b. A rapidly paced rhythm resembling ventricular tachycardia ("runaway pacemaker") is usually due to battery depletion or circuitry malfunction.
3. Specific problems
   a. Failure to pace is detected clinically by the absence of pacemaker spikes in a patient whose intrinsic cardiac rhythm is slower than the programmed pacemaker rate. Causes include:
      (1) Wire fracture is accompanied by acute onset of symptoms that may be sustained or intermittent. It usually occurs at one of three sites: close to the pulse generator, where the lead enters the vein, or within the heart where the lead makes a sharp bend. The chest radiograph will demonstrate lead placement and may also reveal wire fracture.
      (2) Battery depletion is rare if the patient is being monitored often enough.
      (3) Oversensing (sensing electrical events not associated with atrial or ventricular depolarizations) suppresses impulse generation in pacemakers in the inhibit mode. It is more common in patients with unipolar leads and the most common cause of failure to pace.
   b. Failure to sense or capture is detected clinically by the presence of pacemaker spikes occurring at the wrong time (failure to sense) or the presence of pacemaker spikes without associated QRS complexes (failure to capture). Causes include:
      (1) Lead malposition
         (a) Lead displacement usually occurs in the first month after implantation.
         (b) Cardiac perforation usually occurs within 4 days of insertion.
      (2) Wire or insulation fracture
      (3) Battery depletion
      (4) Increased myocardial threshold due to
         (a) Fibrosis or inflammation at the electrode tip
         (b) Lead displacement
         (c) Metabolic and physiologic causes
            i. Metabolic acidosis

ii. Hypoxia
iii. Hyperkalemia
iv. Antidysrhythmic drugs (particularly ones that prolong the QRS)
v. Ischemia
vi. MI

(5) Undersensing (ie, voltage of the patient's intrinsic QRS complex is too low for the pacemaker to sense) is more common with bipolar pacemakers. Sensitivity may be increased by converting to a unipolar pacemaker.

(6) Oversensing (in pacemakers in the triggered mode): pacemaker spikes appear when none is expected because the pacemaker senses and reacts to events other than true cardiac events.

c. Pacemaker-mediated tachycardia

(1) Occurs only in patients with dual-chamber pacemakers that are programmed for synchronous atrioventricular pacing

(2) Requires the presence of ventriculoatrial conduction; is a form of reentrant tachycardia

(3) Can be precipitated by a premature atrial contraction or a premature ventricular contraction

(4) Management

(a) May be terminated by using a pacemaker magnet to briefly turn off the sensing function

(b) Definitive therapy requires reprogramming of the atrial refractory period by a pacemaker specialist.

d. Runaway pacemaker

(1) May be triggered by battery depletion

(2) Rarely occurs today because most newer pacemakers have built-in safety circuits.

(3) Heart rate is frequently >200 beats per minute.

(4) Management

(a) Placement of a pacemaker magnet over the pacemaker may convert the pacemaker to the magnet rate and break the tachycardia.

(b) If this is unsuccessful, and the patient is hemodynamically unstable, the pacemaker must be disconnected. To do this, exteriorize the pacer and cut the electrode wires. These wires may be reconnected to a temporary pacer if the patient's underlying rhythm is unstable.

V. AUTOMATIC IMPLANTABLE CARDIOVERTER DEFIBRILLATORS (AICDs)

A. Indications

1. Placed in patients at high risk of fatal dysrhythmias (ventricular tachycardia, ventricular fibrillation) and sudden cardiac death
2. Included in this high-risk group are patients who survived an episode of sudden cardiac death and those with a prior MI or Brugada syndrome; in these patients, AICDs decrease the risk of sudden cardiac death remarkably from 30%–45% per year to <2% per year.

B. AICD issues in the emergency department

1. Device ineffectiveness may result from lead fracture (sometimes detectable on radiograph) or failure of one of the other components of the device (eg, battery depletion); malfunctions have also been reported from strong electromagnetic fields and interference from appliances and security/antitheft devices.
2. Frequent and recurrent AICD discharge
   a. May be reported or result from
      (1) More frequent episodes of ventricular fibrillation/ventricular tachycardia
      (2) Sensing malfunction/false sensing (eg, sensing and shocking of supraventricular tachycardia or muscular contractions)
      (3) "Ghost shocks" (patient reports shocks when none have occurred)
   b. Patient evaluation
      (1) Continuous cardiac monitoring
      (2) ECG and chest radiograph
      (3) Cardiac markers and drug levels
      (4) Potassium, magnesium, and calcium levels (hypomagnesemia is a common cause of dysrhythmias in AICD patients)
      (5) Telemetry interrogation of the AICD
      (6) Consultation with the patient's cardiologist
   c. Inactivation: placement of a magnet over the AICD generator will inactivate it and thereby prevent further shocks (see below)
3. Performance of CPR and defibrillation with an AICD in place
   a. CPR is performed in the usual manner; while the provider may perceive an AICD shock, it is neither uncomfortable nor dangerous.
   b. External transthoracic defibrillation may also be performed in the standard manner. The paddles should not be placed close to the AICD generator; paddle placement is otherwise unchanged. After successful external cardioversion/defibrillation, the AICD should be tested to confirm that sensing and therapy parameters have not been altered.
4. AICD inactivation: AICDs are generally inactivated by the presence of a magnet; however, some device-to-device variability exists.
   a. With second-generation AICDs, the placement of a donut-shaped magnet over the upper right quadrant of the pulse generator for 30 seconds will inactivate the antitachycardic pacing and shock therapy components of the AICD. Reapplication of the magnet for 30 seconds reactivates it.
   b. With the newer third-generation AICDs, placement of the magnet over the pulse generator inactivates antitachycardic pacing therapy and shocks for as long as the magnet remains in place over the AICD. Removal of the magnet reactivates the device.
5. Infection: Patients who present with erythema, induration, or drainage at the generator site require hospitalization for IV antibiotics. Early infections are usually caused by *Staphylococcus* spp.

ACUTE CORONARY SYNDROME

I. DEFINITION

A. A continuum or progression of coronary artery disease from myocardial ischemia to infarction to necrosis

B. Stable angina → unstable angina → acute MI

II. CLINICAL PRESENTATIONS, RISK FACTORS, AND PREDICTIVE FACTORS

A. Classic presentations of acute coronary syndrome

1. Stable angina
   a. Transient, episodic chest discomfort that is predictable and reproducible, ie, familiar symptoms occur from a characteristic stimulus that improve with rest or sublingual nitroglycerin within a few minutes
   b. These patients are usually sent home or observed briefly in the emergency department.
2. Unstable angina
   a. Angina that is new in onset, occurs at rest, or is similar but somewhat "different" than previous episodes, and is severely limiting or lasts longer than a few minutes. Other signs are an increased frequency of attacks or resistance to prescribed medications that previously relieved the symptoms (eg, nitroglycerin, aspirin).
   b. These patients are admitted for observation or coronary care.
3. Acute MI
   a. Classically, chest discomfort of >15 minutes duration associated with dyspnea, diaphoresis, light-headedness, palpitations, nausea, and/or vomiting. Pain radiation to the inner aspect of one or both arms, shoulders, neck, or jaw is not uncommon and increases the probability that the pain/pressure is ischemic in origin.
   b. Classified as non-ST-segment elevation myocardial infarction (NSTEMI) or ST-segment elevation myocardial infarction (STEMI)
   c. These patients are admitted to a cardiac care unit after appropriate treatment, including reperfusion therapy if needed.

B. Atypical presentations (more common in the elderly, diabetic patients, and women)

1. Chest pain or discomfort (not substernal chest pain/pressure) with or without any of the classic associated symptoms; a history of angina is often absent.
2. Epigastric discomfort/indigestion or nausea and vomiting (may be the only complaint in women or those with an inferior wall MI)
3. Shortness of breath
4. Syncope or confusion
5. Fatigue, dizziness, or generalized weakness
6. Women with prodromal symptoms (unusual fatigue, sleep disturbances, shortness of breath) for a month or more

7. No chest or abdominal pain or discomfort; associated symptoms may or may not be present; patient frequently has vague complaints ("silent" MI).
   a. Approximately 12.5% of all MIs
   b. The following patients should be suspect:
      (1) The elderly
      (2) Diabetic patients
      (3) Those with spinal cord injuries or disease
      (4) Alcoholic patients
      (5) Hypertensive patients or those with hypotensive insults
      (6) Postoperative patients receiving analgesics and postcoronary artery bypass graft patients; these patients generally have a worse prognosis than those with a classic presentation.

C. Risk factors
   1. Major
      a. Cigarette smoking
      b. Hypertension
      c. Diabetes mellitus
      d. Hypercholesterolemia
      e. Family history of coronary artery disease before age 55 in a first-degree relative
      f. Previous history of coronary artery disease, peripheral vascular disease, hypercoagulability, or carotid arteriosclerosis
   2. Other
      a. Male sex
      b. Advanced age
      c. Methamphetamine use
      d. Cocaine use (especially within an hour of presentation or when combined with ingestion of ethanol → cocaethylene, a longer-acting and more toxic metabolite of cocaine)
      e. Obesity
      f. Inactive lifestyle
      g. Postmenopausal state
      h. Inflammatory disorders (eg, lupus)

D. Predictive factors of an evolving acute MI (greater clinical significance than risk factors)
   1. Prior history of ischemic heart disease
   2. Chest pain/discomfort that is worse than usual angina
   3. Pain that is similar to that of a prior acute MI, lasts longer than an hour, or radiates to the left shoulder/arm

III. DIAGNOSIS

A. History

1. A thorough history is the most important tool for identifying patients with cardiac ischemia and is adequate by itself to initiate hospital admission.
2. Patients with a suspicious history and normal initial ECG and cardiac enzymes should be admitted to the hospital or chest pain evaluation/observation unit with a diagnosis of "exclude MI"; typically, <25% of these patients will have a diagnosis of "acute MI" on discharge.
3. A patient without chest pain whose initial ECG and cardiac enzymes are normal may still have an MI.

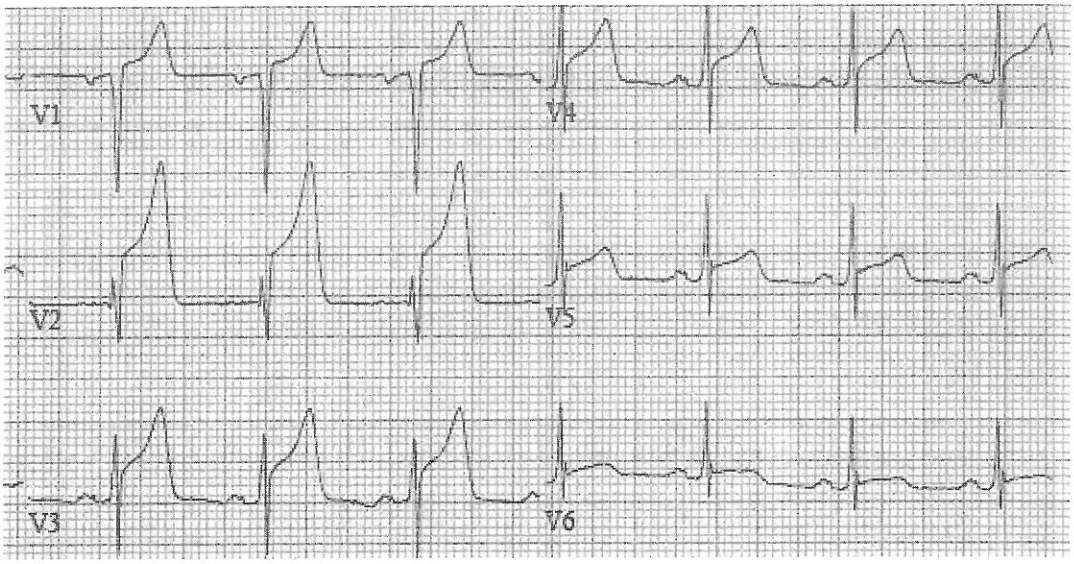
B. Response to medication: clinical improvement after administration of nitroglycerin or an antacid (eg, "GI cocktail") does not exclude an acute MI.

C. ECG

1. **Single most important adjunctive diagnostic test for assessing patients suspected of having acute myocardial ischemia. An ECG should be obtained and reviewed within 10 minutes of patient's arrival in emergency department. The initial ECG is diagnostic, however, in only 25%–50% of patients presenting with an acute MI. Therefore, the finding of a normal, nondiagnostic, or nonspecific ECG in a patient with chest pain does not exclude acute coronary syndrome.**
   a. **ECG findings suggestive of the presence of an acute STEMI include the presence of ST-segment elevation, Q waves, and inverted T waves. Increased amplitude of the R and T waves ("giant" R waves and "hyperacute" T waves) is actually the first change to occur in an evolving MI, but this finding is transient and has usually resolved by the time the patient arrives in the emergency department.**
   b. **ST-segment elevation (representing the current injury) is usually the earliest sign to be recorded and may be accompanied by associated reciprocal changes. The initial upsloping portion of the ST segment is usually convex or flat (horizontally or obliquely) in STEMI; exceptions, however, do occur. This is followed over the next few hours by the development of Q waves (representing myocardial necrosis but not the severity of the infarct) and, finally, T wave inversion.**
      (1) **Isolated ST segment elevation (no Q waves or T wave changes) may occur in the absence of acute coronary syndrome (especially if it does not occur in characteristic leads); this is seen most often with ECG evidence of left ventricular hypertrophy (most common) and left bundle-branch block; however, a new left bundle-branch block in this setting may indicate acute coronary syndrome. An isolated ST-segment elevation may also occur with a ventricular aneurysm.**
      (2) **Q waves without associated ST and T wave changes may be due to an old (not new) MI in the presence of left bundle-branch block; however, Q waves in leads I, aVL, V_5, and V_6 suggest acute MI.**
      (3) **Isolated T-wave inversion (no Q waves or ST changes) indicates ischemia (not acute infarction).**
   c. **Reciprocal changes: ST segment depression (horizontal or downsloping) that occurs in leads opposite to those with ST-segment elevation. The presence of these changes increases the positive predictive value of the ECG diagnosis of STEMI to >90% and denotes a patient at higher risk of later complications.**

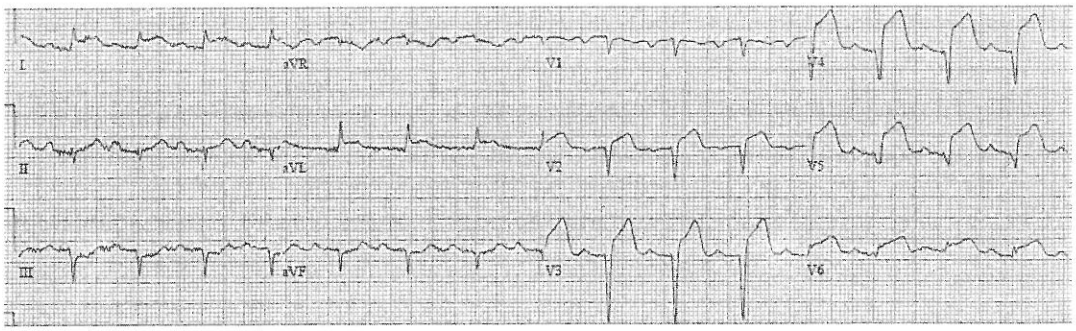
d. ECG localization of the site of infarction

(1) Acute anterior wall MI (left anterior descending): ST-segment elevation in leads I, aVL, and V_1–V_4 (segment depression in leads II, III, and aVF) represents a greater risk of development of conduction abnormalities (AV blocks, bundle-branch blocks, fascicular, and infranodal blocks) and left ventricular dysfunction (CHF)



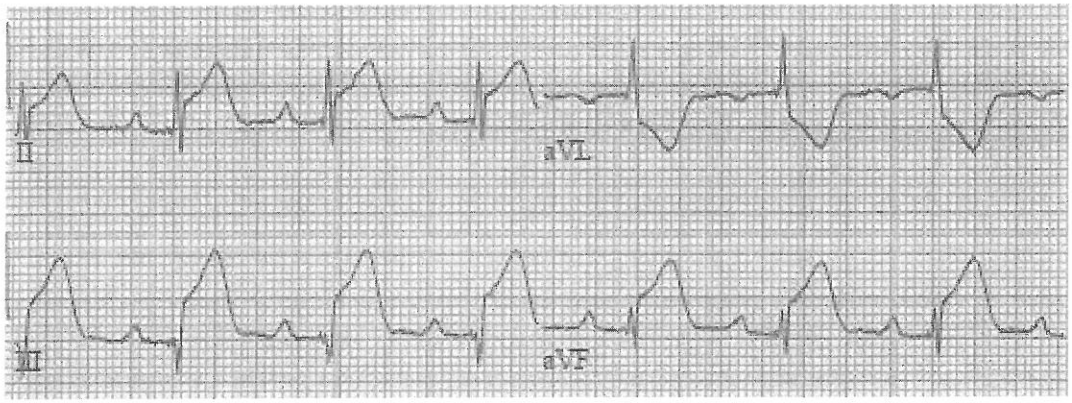
Courtesy of Dr. Michael McCrea

(2) Acute lateral wall MI (left anterior descending or left circumflex artery): ST segment elevation in leads I, aVL, and V_5–V_6 with reciprocal ST-segment depression in leads V_1, V_{3R}, and V_{4R} represents a risk of development of left ventricular dysfunction.



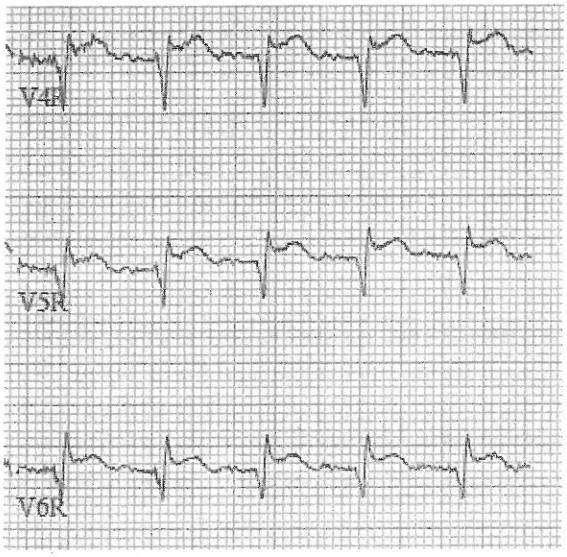
Courtesy of Dr. Michael McCrea

(3) Acute inferior wall MI (right coronary artery): ST-segment elevation in leads II, III, and aVF with reciprocal ST depression in I, aVL, and V_1–V_4 represents a greater incidence of increased vagal tone–mediated dysrhythmias such as sinus bradycardia and varying degrees of AV block.



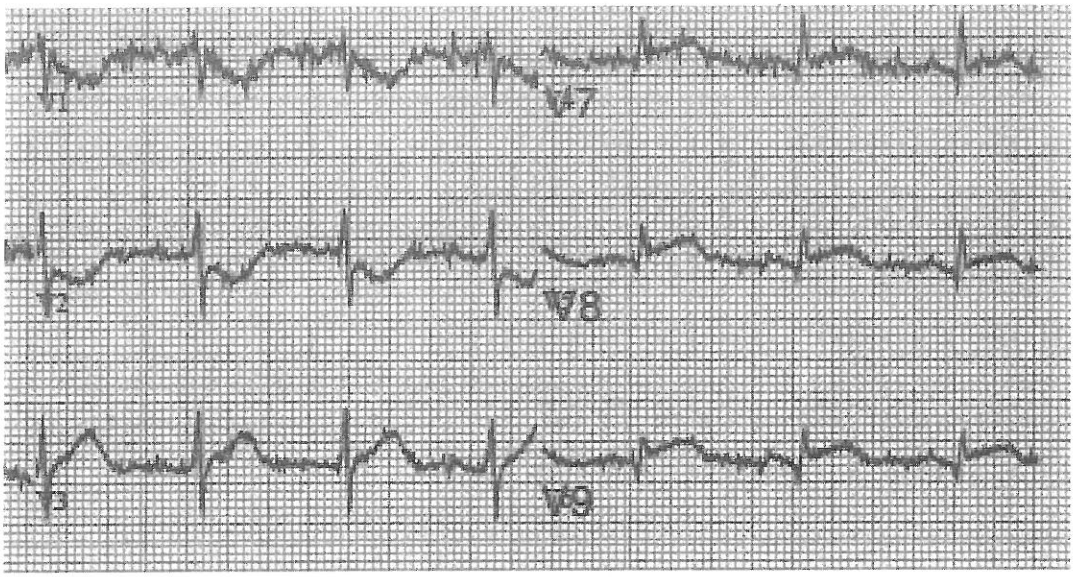
Courtesy of Dr. Michael McCrea

(4) Right ventricular wall MI (RCA): ST-segment elevation in leads V_{3R} and V_{4R} represents a greater risk of developing hypotension and cardiogenic shock. Most right ventricular wall MIs occur in association with an inferior wall MI; ST elevation will be mild.



Courtesy of Dr. Michael McCrea

(5) Acute posterior wall MI (circumflex artery or posterior descending artery off right coronary artery): Because none of the "routine" ECG leads face the posterior surface of the heart, the presence is usually inferred from the finding of reciprocal changes in the anterior leads, particularly V_1 and V_2; ST-segment depression in association with abnormally tall R waves in these leads is the main ECG feature of this diagnosis. When a posterior wall MI is suspected, obtain "additional" posterior leads (V_7, V_8, and V_9); the presence of ST-segment elevation in these leads confirms the diagnosis.



Courtesy of Dr. Michael McCrea

e. Additional lead ECGs

(1) Allow more accurate characterization of acute inferior wall MIs by improving identification of associated right ventricular and posterior wall involvement

(2) Incorporate additional leads to better visualize the right ventricle (V_{4R}) and posterior wall of the left ventricle (V_7, V_8, and V_9), areas that are poorly defined on standard 12-lead ECGs.

(3) These ECGs should be obtained in patients being admitted for suspected acute ischemia who have:

(a) ST-segment depression or suspicious isoelectric ST segments in leads V_1–V_3 (posterior infarct)

(b) T-segment elevation in leads II, III, and aVF (inferior wall infarct) → screen for right ventricular infarction as well (V_{4R}).

(c) Isolated ST-segment elevation in V_1 or ST segment elevation in leads V_1 and V_2 (right ventricular infarct)

(d) Borderline ST-segment elevation in leads II, III, and aVF or in leads V_5 and V_6 (inferior wall infarct)

(e) Symptoms consistent with right ventricular ischemia (eg, epigastric pain, significant hypotension after administration of nitroglycerin)

(4) Clinical significance

(a) Patients with an acute posteroinferior wall MI or right ventricular infarction usually have larger-sized MIs, lower ejection fractions, and higher morbidity/mortality rates.

(b) Associated hypotension in patients with right ventricular infarction will likely respond to IV fluids; morphine, nitrates, and diuretics may further compound the situation.

(5) Use of additional lead ECGs should be restricted to the patient population described above, ie, those patients with suspected posterior to right ventricular infarction. Obtaining additional lead ECGs in all emergency department patients with chest pain does not appear to have therapeutic or diagnostic benefits, and results in an unacceptable increase in false-positive diagnoses.

2. The initial ECG is also useful in the following situations:
   a. Screening for nonischemic (but potentially serious) causes of chest pain, such as pericarditis and pulmonary embolism.
   b. Stratifying the risk of an adverse outcome in association with in-hospital disposition
      (1) Patients at high risk of complications and death should be admitted to a cardiac care unit or ICU. Included in this group are patients with any of the following ECG findings:
         (a) Ischemic ST or T-wave changes in contiguous leads
         (b) Pathologic Q waves (>0.04 seconds long or 25% of R-wave height)
         (c) Left ventricular hypertrophy
         (d) Left bundle-branch block (new or age uncertain)
         (e) Right bundle-branch block and ST changes
         (f) Paced rhythm
      (2) Patients at low risk of complications can safely be admitted to a step-down unit. Included in this group are patients whose initial ECG is either normal or have nonspecific ST–T wave changes.
   c. Establishing the criteria that determine which therapeutic interventions will be used (ß-blockers, thrombolytic therapy, etc)
3. Serial ECGs
   a. Indicated in patients with nondiagnostic ECGs in whom there is concern of possible ongoing ischemia
   b. Capture ischemic ECG changes, demonstrate ECG stability, and detect silent ischemia; ST segment trend monitoring may improve detection.
   c. Patients with normal or nonspecific initial ECGs and subsequent confirmed acute MIs within 72 hours still have a high rate of mortality and life-threatening complications.

D. Serum markers of acute MI

1. Myoglobin starts increasing within 2–3 hours of infarction and peaks within 4–24 hours. It is more sensitive than the total creatine kinase (CK) and CK-MB but is not specific for cardiac muscle and, therefore, has a high false-positive rate. Therefore, it cannot reliably identify or exclude a MI at any time junction; it is best used in conjunction with other markers.
2. CK starts increasing within 4–6 hours of infarction, peaks within 12–24 hours, and returns to baseline in ~48 hours. However, it is not specific for an acute MI; in addition to heart muscle, it is also found in skeletal muscle and brain tissue, so increases can be due to disorders of any of these organ systems.
3. CK-MB starts increasing within 4–6 hours of infarction, peaks within 20 hours, and is more specific than CK (but still not nearly 100% specific for MI). The newer monoclonal

assays that directly measure CK-MB mass (and subforms) have essentially replaced the old electrophoretic assays, because they are more sensitive and faster to run.

4. Troponin (TN) T and I: appear in the serum within 3–4 hours of infarction, peak in 12–24 hours, and remain increased for 1–2 weeks. The newest-generation TN assays detect TN release within 1–2 hours of infarction. Troponin I is the most specific cardiac marker available (>90%). Troponin T is not as specific as Troponin I (but far more than CK-MB) and is valuable in predicting cardiovascular complications in unstable angina and acute MI.
5. Appropriate use of serum markers
   a. The decision to admit or discharge a patient should be based primarily on the patient's history and clinical presentation, not on the presence or absence of increased cardiac markers.
      (1) Detection of these markers requires that sufficient myocardial cell damage has occurred and that enough time has passed for these markers to be released into the serum.
      (2) Initial determination of these markers has a low sensitivity for detecting ischemia and cannot be used to reliably diagnose or exclude the presence of acute coronary syndrome.
      (3) No single determination of one serum biomarker of myonecrosis reliably identifies or excludes acute MI in <6 hours of symptom onset.
   b. The laboratory result reported for each serum marker (Table 4) is not as valuable as the pattern of the results as well as an accurate history of the onset of signs and symptoms in a patient with a nondiagnostic ECG.
      (1) When did the signs/symptoms begin (today and before today)?
      (2) What events have occurred in the past 2 weeks that are new or different (changes in medications, dosages, habits, activities, other illnesses, or complaints)?
      (3) Pinpointing the time when things changed and signs/symptoms appeared allows you to plot the timing of enzyme activity and interpret the results more accurately.

Table 4: Cardiac Serum Markers

Serum Marker	Increases (hr)	Peaks (hr)	Remains High (days)
Myoglobin	2–3	4–24	<1
CK	4–8	12–24	<3–4
CK-MB	4–10	20*	<2
Troponins (T + I)*	6	12–18	Troponin I: 7–10 Troponin T: 10–14

*The markers of choice, especially in delayed presentation; their presence is proof a problem exists.

E. Exercise treadmill testing

1. Initial recommended test of choice by the American Heart Association (AHA) for those patients who are able; limited by exercise intolerance and baseline respiratory or orthopedic problems in many patients.
2. Looks for diagnostic ECG changes with exercise

F. Two-dimensional echocardiography

1. Effective in detecting regional wall motion abnormalities that occur in association with an acute MI but is unable to distinguish between ischemia, acute infarction, and an old infarction.
2. Operator-dependent; not readily available
3. In patients with normal resting wall motion, diagnostic utility of echocardiography may be improved when combined with stress testing (ie, "stress echo")
   a. Exercise treadmill if able to exercise per protocol
   b. Chemical, eg, dobutamine, in appropriate patients

G. Radionuclide scanning

1. Technetium 99m sestamibi imaging
   a. Technetium 99 is a myocardial perfusion tracer that is taken up by the myocardium in proportion to blood flow; sestamibi is a small protein that is labeled with radiopharmaceutical technetium 99.
   b. It can detect perfusion defects and segmental wall abnormalities such as hypokinesis.
   c. In patients with active chest pain and a nondiagnostic ECG, it has nearly 100% sensitivity and 83%–92% specificity; in pain-free patients, however, its sensitivity is only 65%.

H. Chest pain evaluation unit

1. A safe, effective alternative to routine admission of low-intermediate risk patients with chest pain
2. Protocols vary but usually involve serial studies (ECGs, markers) and selective stress testing for evaluation based on risk stratification.

IV. MANAGEMENT

A. IV (normal saline), oxygen (2–4 L/min), cardiac monitor, pulse oximeter

B. Antiplatelet agents

1. **Aspirin**
   a. **Beneficial (through its antiplatelet activity) in the management of angina (reduces the risk of acute MI) and infarction; decreases mortality, infarct size, and rate of reinfarction associated with unstable angina and acute MI whether or not thrombolytic therapy is given. Maximum benefit occurs if given in the first 4 hours of onset of chest pain.**
   b. **Irreversibly acetylates platelet cyclooxygenase; has a rapid onset of action (within 60 minutes)**
   c. **Unless contraindications exist, administer 325 mg on arrival in the emergency department and continue indefinitely. It should be chewed so as to maximize bioavailability. If vomiting is present, it may be given as a rectal suppository.**
2. **Platelet receptor inhibitors**
   a. **Available agents: clopidogrel, prasugrel, ticagrelor**
   b. **Mechanism of action: affect platelet activity (including aggregation) through inhibition of ADP platelet activation**

c. Administer in the emergency department only as a substitute for aspirin in patients with true aspirin allergy.

d. STEMI: loading dose of one of these agents should be given as early as possible or at the time of percutaneous coronary intervention (PCI). Not administering until the coronary anatomy is defined at catheterization prevents possible delay in coronary artery bypass graft (CABG) if needed because of prolonged antiplatelet effects of these agents.

e. Unstable angina/NSTEMI: administer a loading dose of one of these as early as possible if there is planned early intervention, or begin maintenance dose at time of admission if delayed or no intervention is planned.

f. Prasugrel is *contraindicated* in patients with any prior history of transient ischemic attack or stroke because of increased risk of intracranial hemorrhage. Prasugrel should not be administered in the emergency department for unstable angina/NSTEMI; its use should be deferred to the time of PCI.

3. Glycoprotein IIb/IIIa receptor antagonists

a. Available agents include abciximab, eptifibatide, and tirofiban.

b. Mechanism of action: in the presence of platelet activation, the glycoprotein IIb/IIIa receptor inhibitors block the final common pathway for platelet aggregation.

c. For both STEMI and unstable angina/NSTEMI, IIb/IIIa inhibitors may be used as adjunctive therapy and started either before or at the time of catheterization for PCI but do not have to be started in the emergency department.

C. Anticoagulant (antithrombotic) therapy

1. Unfractionated heparin (UFH)

a. Indicated for all patients presenting with acute coronary syndrome: STEMI, NSTEMI, and high-risk unstable angina

b. Acts as an indirect thrombin inhibitor by accelerating the action of antithrombin III (a thrombin inhibitor) and activated factors IX, X, and XI. Its antithrombotic activity complements the antiplatelet activity of aspirin to prevent progression of ischemia to acute MI; when used together, heparin and aspirin are more effective than either used alone.

c. Beneficial effects

(1) In the acute MI setting, it decreases the incidence of deep-vein thrombosis, reinfarction, nonhemorrhagic stroke, and the formation (as well as embolization) of left ventricular thrombus.

(2) When administered to patients receiving thrombolytic agents, it prevents/decreases the incidence of reocclusion.

(3) In patients with unstable angina, it may be useful in decreasing the rate of subsequent transmural infarction.

(4) It may be preferred by cardiologists who are taking patients to the catheterization laboratory, because it can be turned off.

d. Recommended as the anticoagulant of choice by the AHA for STEMI patients undergoing PCI. Low-molecular-weight heparin (LMWH) is no longer recommended for STEMI in the 2013 AHA STEMI update.

e. Dosage: bolus of 50–70 units/kg (maximum 5,000 units) followed by an initial infusion of 12 units/kg/hr (maximum 1,000 units/hr) adjusted to maintain a partial thromboplastin time 1.5–2 times control (50–70 seconds)

2. **LMWHs may be considered acceptable alternative to UFH in patients <75 years old without significant renal dysfunction**
   a. **Produce less bleeding than UFH with equivalent or better antithrombotic effects**
   b. **Although LMWHs are more expensive than UFH, some argue that the overall cost of their use is similar to or less than that of UFH, because serum monitoring is not necessary.**
   c. **For patients with planned CABG within 24 hours, UFH is now recommended over enoxaparin.**
   d. **Enoxaparin is equally recommended with UFH in patients with unstable angina/ NSTEMI in the absence of renal failure**
3. **Direct thrombin inhibitors**
   a. **Bivalirudin directly inactivates thrombin**
   b. **Bivalirudin may be used as a primary anticoagulant alternative to or in conjunction with UFH in patients undergoing PCI for STEMI.**
   c. **Bivalirudin may also be selected as initial anticoagulant for unstable angina/NSTEMI patients. However, in those patients, who then require PCI or CABG, bivalirudin should be stopped and the patient switched to UFH.**
4. **Direct factor Xa inhibitor**
   a. **Selectively binds to antithrombin III, which potentiates factor Xa activation and thus inhibits thrombin formation**
   b. **Fondaparinux is preferred to LMWH in patients with renal dysfunction.**
   c. **Fondaparinux must be combined with UFH to avoid catheter-related thrombosis in patients undergoing PCI.**

D. Nitroglycerin

1. Decreases ischemic pain (and consequently catecholamine release); no proven reduction in mortality but recommended nevertheless if no contraindications
2. Pathophysiology
   a. Dilates collateral coronary vessels and increases collateral blood flow to ischemic myocardium
   b. Has antiplatelet effects and reduces infarct size and mortality
   c. Decreases myocardial oxygen demand by decreasing preload, left ventricular end-diastolic volume, and afterload (which makes it the drug of choice if coexisting left ventricular failure)
   d. May also reduce myocardial susceptibility to ventricular dysrhythmias during ischemia and reperfusion
3. Should be administered to patients with ischemic chest pain whose systolic blood pressure is >90 mmHg. Start with sublingual nitroglycerin 0.4 mg every 3–5 minutes as needed for pain. Follow with an infusion of 10–20 mcg/min and increase by 5–10 mcg/min every 5–10 minutes until pain is controlled or systolic blood pressure is decreased by 10%.
4. Adverse reactions
   a. Hypotension usually responds to a fluid bolus and leg elevation; it is particularly common when nitroglycerin is given to patients with inferior wall MIs with associated right ventricular infarction as well as to those with hypovolemia or bradycardia; caution should be exercised when administering nitroglycerin to these patients.
   b. Reflex tachycardia may be moderated by the concomitant use of ß-blockers.

c. Administration of nitroglycerin to patients taking sildenafil citrate or other longer-acting medications for erectile dysfunction is contraindicated. In these patients, nitroglycerin can precipitate a sudden and profound decrease in blood pressure, which leads to decreased coronary perfusion and progression of myocardial ischemia to infarction with all of its potential consequences. Nitroglycerin should be avoided for 12–24 hours after using medications for erectile dysfunction.

E. β-Blockers

1. The routine use of early IV ß-blockers for patients presenting with acute coronary syndrome is associated with an increased risk of cardiogenic shock without a significant decrease in mortality. Consequently, IV ß-blockers are indicated only in patients with acute coronary syndrome who also demonstrate tachydysrhythmias (eg, rapid atrial fibrillation) or who have intractable hypertension despite nitrates.
2. ß-blockers should be started orally within the first 24 hours of admission in patients who have none of the following:
   a. Signs of heart failure
   b. Evidence of low-output state
   c. Risk factors for cardiogenic shock
      (1) Age >70 years old
      (2) Systolic blood pressure <120 mmHg
      (3) Sinus tachycardia >110 beats per minute or heart rate <60 beats per minute
   d. Other relative contraindications and precautions to ß-blockade, including:
      (1) PR interval >240 milliseconds
      (2) Second- or third-degree AV block
      (3) Active asthma or reactive airway disease
      (4) Cocaine toxicity
      (5) Acute right ventricular infarction
3. Unless contraindications exist, administer the following:
   a. Metoprolol 50–100 mg orally *or*
   b. Atenolol 50 mg orally *or*
   c. Labetalol 10–20 mg IV if blood pressure control is needed; may repeat dose or double the dose as needed in 20–30 minutes *or*
   d. Esmolol 500 mcg/kg IV (over 1 minute) followed by an infusion of 50 mcg/kg/min titrated to a maximum dosage of 200 mcg/kg/min if rate control of a tachydysrhythmia is needed
4. Pathophysiology
   a. Decrease myocardial oxygen demand by decreasing heart rate and myocardial contractility
   b. Increase coronary blood flow by increasing time of diastole and decreasing myocardial wall tension
   c. Markedly decrease rate of myocardial rupture (particularly in the elderly)
   d. Decrease incidence of ventricular fibrillation and cardiac rupture
   e. Decrease platelet aggregation

F. Morphine

1. Should be administered if chest pain persists despite adequate management with antiplatelet, anticoagulant, and anti-ischemic agents
2. Pathophysiology: decreases pain and anxiety → decreases circulating catecholamines → decreases tendency toward dysrhythmias and myocardial oxygen demand
3. Administer 2–4 mg every 5–10 minutes as needed for pain.
4. Adverse effects
   a. Hypotension and bradycardia: respond to fluid bolus and atropine
   b. Do not administer to patients with a heart rate <50 beats per minute or to those with symptomatic hypotension.
   c. Respiratory depression responds to naloxone.

G. Reperfusion therapy

1. **Percutaneous coronary intervention (PCI)**
   a. **The preferred management for STEMI over thrombolytic therapy in all patients**
      (1) **Appears to be more effective than thrombolysis in opening occluded arteries, because it can treat the underlying fixed obstructed coronary artery lesion as well as relieve the acute thrombosis.**
      (2) **Associated with a lower incidence of recurrent ischemia, reinfarction, intracranial hemorrhage, and death**
      (3) **Results in similar left ventricular function**
      (4) **Also has the advantage of allowing the operator to define the site and extent of coronary artery stenosis if needed for CABG**
   b. **Not available on a 24-hour basis in many institutions**
   c. **The AHA 2013 recommendations for STEMI and PCI versus thrombolytics are:**
      (1) **If a patient presents to a primary PCI hospital, first-medical contact to device time should be <90 minutes. Note: it is no longer "door-to-balloon," ie, time starts with prehospital personnel contact if patient arrives by EMS.**
      (2) **If a patient presents to a non-PCI hospital and transfer and PCI can be performed with a first-medical contact to device time of <120 minutes, immediate transfer for primary PCI is the recommended management strategy with goal of "door-in, door-out" time at the transferring hospital of ≤30 minutes.**
      (3) **If a patient presents to a non-PCI and transfer for PCI cannot be assured within 120 minutes, thrombolytics should be administered within 30 minutes of patient arrival to the non-PCI hospital emergency department.**
2. **Thrombolytic (fibrinolytic) therapy**
   a. **Agents**
      (1) **Tissue plasminogen activator**
      (2) **Alteplase**
      (3) **Reteplase**
      (4) **Tenecteplase**
   b. **These agents convert plasminogen to plasmin (its active form), which in turn lyses the fibrin content of acute intracoronary thromboses → reperfuses coronary arteries → reduces infarction size, improves residual left ventricular function, and increases survival. The fibrin specificity of these agents varies.**

c. If thrombolytic therapy is chosen for reperfusion for STEMI, it should be administered within 30 minutes of patient's arrival to the emergency department.

d. Agent availability and administration protocols vary from hospital to hospital.

e. American Heart Association/American College of Cardiology criteria for thrombolysis

(1) Class I (treatment benefit has been established): ST elevation >0.1 mV in two or more contiguous leads with ischemic symptoms of <12 hours duration

(2) Class IIa (likely benefit): evidence of ongoing ischemia of 12–24 hours with either large area of myocardium at risk or hemodynamic instability

(3) Class III (harmful, not indicated) for ST depression unless:

(a) Suspected isolated posterior MI

(b) Associated with ST elevation in aVR, which signifies either triple vessel disease occlusion or left main coronary artery occlusion

f. Absolute contraindications to thrombolysis

(1) Any prior cerebral hemorrhage

(2) Known structural CNS lesion (AV malformation, tumor, etc)

(3) Ischemic stroke within 3 months (unless ischemic symptoms of <3 hours)

(4) Significant closed head or facial injury within 3 months

(5) Suspicion of aortic dissection

(6) Active bleeding (excluding menses) or bleeding disorders

g. Relative contraindications

(1) History of chronic, severe, poorly controlled hypertension or severe hypertension on admission (systolic blood pressure >180 mmHg or diastolic blood pressure >110 mmHg)

(2) Traumatic or prolonged (>10 minutes) CPR

(3) Noncompressible vascular punctures

(4) Major surgery or internal bleeding within 3–4 weeks

(5) Any other CNS disease (structural or functional, eg, dementia) not noted above

(6) Pregnancy

(7) Active peptic ulcer

(8) Current use of anticoagulants (the higher the INR, the greater the risk of bleeding)

(9) Prior exposure/allergic reaction to streptokinase or anistreplase (if using these agents)

h. Complications

(1) Systemic bleeding: incidence 2%–10%; usually occurs in the setting of invasive procedures.

(2) Cerebral hemorrhage: incidence <1% (greater with tissue plasminogen activator); associated with high mortality rate (48%)

(3) Hypotension: incidence 3%–10%; more common with streptokinase and anistreplase than tissue plasminogen activator; usually responds to fluid bolus and temporary reduction of infusion rate

(4) Allergic phenomena: incidence 1.5%–2%; usually minor (anaphylaxis is rare); most common with older streptokinase and APSAC

(5) Failure to open occluded coronary arteries (incidence 20%)

H. Intravenous magnesium

Current recommendations are that magnesium should not be given routinely to patients who undergo reperfusion therapy. While it is possible that magnesium is of benefit (particularly in patients not receiving perfusion therapy), further studies are required to evaluate its use in the unstable angina/acute MI setting.

I. ACE inhibitors

1. When administered within the first 24 hours, these agents decrease the incidence of severe ventricular dysfunction and death. Unless specific contraindications exist, all patients with an acute coronary syndrome should receive an ACE inhibitor but not until 6 hours after initial therapy has been started in the emergency department and the patient is stable. If given too early, ACE inhibitors can potentiate hypotension.
2. One of the following agents should be administered in the first 24 hours after infarction:
   a. Captopril 12.5 mg every 12 hours orally (a test dose of 6.25 mg may be given)
   b. Lisinopril 5 mg/day orally (if systolic blood pressure is <120 mmHg, use 2 mg/day orally)
3. Contraindications
   a. ACE-inhibitor allergy
   b. Killip Class III or IV heart failure
   c. Hypotension (systolic blood pressure <100 mmHg)
   d. Creatinine >2.5 mg/dL
   e. Renal artery stenosis

J. Glucose-insulin-potassium (GIK) therapy

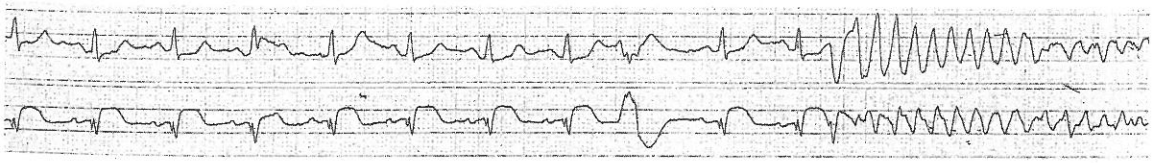
1. Proposed to reduce mortality during an acute MI because of its anti–free fatty acid activity. Free fatty acids are toxic to the ischemic myocardium.
2. The results of a large, controlled, multinational study published in 2005 refuted smaller, earlier studies claiming a reduced mortality rate with GIK therapy. Therefore, GIK therapy is not recommended in the management of acute MI.

V. COMPLICATIONS

A. Myocardial ischemia produces altered electrical depolarization and contractility, which are the major complications of an acute MI (ie, dysrhythmias and left ventricular failure).

B. Dysrhythmias

1. The prehospital phase is the period associated with the highest incidence of lethal dysrhythmias; the occurrence of primary ventricular fibrillation (no evidence of CHF or hypotension) is greatest in the first hour of infarction.



Courtesy of Dr. Michael McCrea

2. Treatment is indicated if the dysrhythmia
   a. Exacerbates myocardial ischemia or
   b. Could potentially deteriorate into cardiac arrest
   c. Treat specific dysrhythmias per current ACLS protocols.
3. Lidocaine is no longer recommended as a prophylactic antidysrhythmic for acute MI or ischemia; its use in this setting has been associated with an increased mortality rate.
4. For premature ventricular contractions occurring in the presence of acute MI, the initial treatment priority should be to optimally manage the underlying ischemia/infarction with oxygen, aspirin, nitroglycerin, and reperfusion therapy (ie, thrombolytics or PCI). Any electrolyte abnormalities should also be corrected. Previously, empiric treatment with ß-blockers was recommended to prevent premature ventricular contractions. However, new evidence refutes this practice, and there is possibly added harm by increasing risk of cardiogenic shock in patients with acute MI.

C. Left ventricular failure (CHF, pulmonary edema, cardiogenic shock)

1. Acute MI → impaired contractility of the left ventricle
   a. Impairment ≥25% of the left ventricle → CHF/pulmonary edema
   b. Impairment ≥40% of the left ventricle → cardiogenic shock
2. Pathogenesis: pump failure = CHF; over time, this leads to increased pulmonary vascular congestion → pulmonary edema. Cardiogenic shock can occur with either acute, severe pump failure or acute, severe pulmonary edema (each of which further impairs left ventricular function).
3. For management, see CHF/pulmonary edema, pages 64–67.

D. Conduction disturbances

1. AV blocks
   a. First-degree AV block and Mobitz Type I second-degree AV block
      (1) Generally due to increased vagal tone
      (2) Rarely progress to complete AV block
      (3) Usually associated with an inferior MI (think Mobitz Type I and Inferior)
      (4) Generally responds to drug therapy (atropine)
   b. Mobitz Type II second-degree AV block
      (1) Generally due to destruction of infranodal conduction tissue
      (2) Sudden progression to complete AV block may occur
      (3) Usually associated with an anterior MI
      (4) Prophylactic pacemaker therapy is indicated
2. Bundle-branch blocks
   a. In general, bundle-branch blocks (old or new) associated with an acute MI identify patients who are more likely to develop CHF, AV block, and ventricular fibrillation.
   b. In patients with an acute anterior wall MI and a new right bundle-branch block, there is a high risk of developing complete AV block and/or cardiogenic shock.

E. Other less common complications of acute MI

1. Free left ventricular wall rupture
2. Ventricular septal rupture
3. Papillary muscle dysfunction/rupture

4. Acute mitral regurgitation
5. Left ventricular aneurysm
6. Thromboembolism
7. Acute pericarditis
8. Most of these complications are delayed in onset, occurring 1 or more days after infarction.

CONGESTIVE HEART FAILURE

I. ETIOLOGY, PRECIPITATING FACTORS, AND SIGNS AND SYMPTOMS

A. Left ventricular failure

1. Ischemic heart disease (most common cause)
2. Idiopathic dilated cardiomyopathy
3. Hypertension
4. Valvular disease (aortic or mitral)
5. High-output states (anemia, thyrotoxicosis, AV fistula, beriberi, Paget disease)
6. Congenital heart disease
7. Coarctation of the aorta

B. Right ventricular failure

1. Left ventricular failure (most common cause)
2. Pulmonary arterial hypertension (and right ventricular failure = cor pulmonale)
3. Valvular disease (tricuspid or pulmonic)
4. Restrictive or infiltrative cardiomyopathies
5. Myocarditis and some forms of congenital heart disease
6. Right ventricular infarction
7. Pulmonary embolism
8. Chronic pulmonary disease

C. Precipitating factors

1. Myocardial ischemia or infarction
2. Noncompliance with medications
3. Tachydysrhythmias (eg, atrial fibrillation) and severe bradydysrhythmias
4. Dietary indiscretion (sodium overload)
5. Administration of drugs that impair cardiac function (ß-blockers or calcium channel blockers) or result in sodium retention (glucocorticoids, NSAIDs, nasal decongestants, vasodilators)
6. Increased hemodynamic demand due to infection, trauma, physical overexertion, environmental stress, or pregnancy
7. Hypoxia (due to pulmonary embolism or pneumonia)
8. Cardiac disease progression (coronary insufficiency and CHF are the most common precipitating causes of acute cardiogenic pulmonary edema)
9. Severe hypertension
10. COPD (the leading cause of chronic cor pulmonale)
11. Acute myocarditis or endocarditis
12. Acute valvular dysfunction

D. Signs and symptoms

1. Shortness of breath (most common symptom)
2. Paroxysmal nocturnal dyspnea/orthopnea
3. Nocturnal angina
4. Moist rales and/or wheezing ("cardiac asthma")
5. Cough
6. Fatigue and/or weakness
7. Pleural effusion (usually right-sided)
8. Tachypnea and tachycardia
9. S_3 gallop (due to reduced left ventricular compliance)
10. S_4 gallop (not heard in patients with atrial fibrillation)
11. Hepatojugular reflux → jugular venous distention (right ventricular failure)
12. Pulsus alternans (alternating strong and weak arterial pulse waveform)
13. Ascites (right ventricular failure)
14. Hepatic enlargement/tenderness (right ventricular failure)
15. Pale, clammy skin or diaphoresis
16. Neck vein distention (right ventricular failure)
17. Anxiety
18. Nocturia
19. Dependent edema (right ventricular failure)

II. DIAGNOSTIC EVALUATION

A. Radiographs

The cardiac silhouette is generally enlarged (although it may be normal size) in all three stages, associated pleural effusions (usually right-sided) are common, and chest radiograph findings may be delayed up to 12 hours relative to symptom onset.

1. Stage 1: pulmonary vascular redistribution to upper lung fields ("cephalization")
   a. Chest radiograph → fullness or prominence of the pulmonary vessels in the apices
   b. Predominant symptom (if any are present) is dyspnea.
2. Stage II: interstitial edema
   a. Chest radiograph → pulmonary vessels enlarged with blurred shadows, Kerley B lines (1-cm horizontal markings at the periphery of lung fields) are also present.
   b. Predominant symptom is dry cough.
3. Stage III: alveolar edema (frank pulmonary edema)
   a. Chest radiograph, → bilateral confluent perihilar infiltrates creating a "butterfly" pattern
   b. Predominant symptom is a wet cough with production of a frothy, pink sputum.

B. Laboratory studies

1. Brain-type natriuretic peptide (BNP) is used as a serum marker for acute CHF.
2. Pathogenesis: cardiac hypertrophy/wall stress and volume overload → release of endogenous BNP by atrial and ventricular myocytes → smooth muscle relaxation and vasodilation, diuresis and natriuresis

3. Acute CHF can lead to high serum levels of BNP, which are especially significant in the absence of pulmonary disease and advancing age (when moderate increases can occur).
4. A serum BNP level <100 pg/mL reliably excludes the diagnosis of acute CHF (sensitivity 90%, specificity 76%).

III. MANAGEMENT

A. Management of acute cardiogenic pulmonary edema

1. First-line therapy for all patients who are not hypotensive
   a. Place the patient in an upright position, attach a cardiac monitor and pulse oximeter, start an IV line, check the vital signs, and review a monitor strip.
   b. Oxygen
      (1) All of these patients are hypoxemic and should immediately be given supplemental oxygen; it is the most important agent in the management of pulmonary edema.
      (2) Administer high-flow 100% oxygen via a nonrebreather mask at 10–15 L/min; maintain adequate arterial saturation.
      (3) Early application of noninvasive positive-pressure ventilation (NIPPV) improves oxygenation and dyspnea and also reduces the likelihood of intubation in hypercapneic patients. It can be applied with a tight-fitting facial mask (continuous positive-airway pressure [CPAP]) or with a nasal mask (bi-level positive-airway pressure [BiPAP]). The latter has the advantage of being able to separately regulate inspiratory and expiratory positive-airway pressures and generally is better tolerated than CPAP.
      (4) Proceed with intubation if the patient is or becomes obtunded, cannot maintain a PaO_2 >60 mmHg despite receiving 100% oxygen, displays a progressive increase in pCO_2, or demonstrates increasing acidosis. Do not wait for arterial blood gases if patient's clinical condition is deteriorating. Positive end-expiratory pressure (PEEP) can then be applied via the endotracheal tube.
      (5) Because positive-pressure ventilation (either NIPPV or endotracheal ventilation) increases intrathroacic pressure, there is a resultant decrease in preload, which can decrease cardiac output and blood pressure. Therefore, monitor blood pressure carefully.
   c. Initial medications
      (1) Nitroglycerin
         (a) At low doses, nitroglycerin is primarily a venodilator and rapidly acts to decrease preload. Its effects on arterial dilation are less profound and are usually associated with much higher doses. Nitroglycerin also acts to increase coronary blood flow by promoting dilation of large epicardial vessels and is, therefore, the vasodilator of choice in the presence of ischemia or an acute MI.
         (b) Administer sublingual nitroglycerin 0.8–1.2 mg every 5–10 minutes (use higher doses if blood pressure is moderately to severely increased) as a loading dose, then start high-dose nitroglycerin IV drip.

(2) Furosemide

(a) Induces diuresis but in patients with acute cardiogenic pulmonary edema, the diuretic effect may be delayed 1–2 hours because increased afterload results in reduced renal blood flow. Premedication with preload- and afterload-reducing medications hastens the diuretic effect of furosemide.

(b) Dosage: 0.5–1 mg/kg IV push over 1–2 minutes (use dosages in the lower range if patient is not taking furosemide chronically; use the higher dosage range if he or she is).

(3) Morphine sulfate

(a) Although traditionally thought to reduce preload in patients with cardiogenic pulmonary edema, invasive studies have not demonstrated any actual central preload-reducing effects. Other medications are clearly superior for preload reduction (eg, nitroglycerin).

(b) The absence of true hemodynamic benefits combined with the potential for adverse effects (eg, respiratory depression, vomiting, urticaria) favors avoidance of its use in patients with cardiogenic pulmonary edema.

2. Second-line therapy: based on systolic blood pressure and presence or absence of clinical shock

a. Systolic blood pressure >100 mmHg (no signs/symptoms of shock)

(1) Nitroglycerin IV at 10–20 mcg/min; titrate the drip upward in increments of 5–10 mcg/min every 5 minutes until the desired effect is attained or blood pressure decreases to <100 mmHg. (A reduction in preload is usually achieved at 50–80 mcg/min.)

(2) Nitroprusside

(a) A potent mixed venous and arteriolar dilator; reduces both preload and afterload, thereby decreasing pulmonary congestion and reducing cardiac output

(b) Usually reserved for patients with a systolic blood pressure >100 mmHg who do not respond to adequate dosages of the standard preload reducers (nitroglycerin, furosemide) or for patients with low output who require controlled afterload reduction

(c) Dosage: 0.1–5 mcg/kg/min IV; invasive hemodynamic monitoring is strongly recommended.

b. Systolic blood pressure 70–100 mmHg

(1) No signs/symptoms of shock → dobutamine

(a) A direct-acting inotropic agent that is effective in increasing cardiac output and decreasing pulmonary capillary wedge pressure

(b) Dosage: 2–20 mcg/kg/min IV (patients on ß-blockers may need the higher dosage)

(c) Hemodynamic monitoring is advisable, because dobutamine may worsen hypotension.

(d) If dobutamine successfully improves cardiac output and blood pressure, a nitroglycerin infusion can be carefully added for preload reduction, as the patient's blood pressure tolerates.

(e) Dobutamine has some vasodilatory effect as well, so some patients will have a reduction in blood pressure. If this occurs, the addition of a vasopressor may be needed.

(2) Signs/symptoms of shock present → dopamine

(a) Indirect inotropic support as an effective pressor agent; the preferred agent when persistent oliguria or shock is present (systolic blood pressure <100 mmHg) and should be administered before venodilating agents and diuretics when hypotension is present on arrival. Dosage: 5–15 mcg/kg/min IV (patients on ß-blockers may need the higher dosage)

(b) Hemodynamic monitoring is advisable.

(c) The vasopressor effect of dopamine increases afterload, but it can actually worsen pulmonary congestion. To counter this adverse effect, the concurrent use of vasodilatory medications (nitroglycerin or nitroprusside) is recommended, as tolerated by the patient's blood pressure.

(d) Dopamine may also be used in combination with dobutamine; dopamine maintains blood pressure, while dobutamine prevents further increase in pulmonary congestion.

c. Systolic blood pressure <70 mmHg with obvious signs/symptoms of shock □ norepinephrine

(1) Potent vasoconstrictor and inotropic agent

(2) Dosage: 0.1–0.5 mcg/kg/min

(3) Hemodynamic monitoring is advisable.

(4) Similar to dopamine, norepinephrine is best used concurrently with a vasodilator, as tolerated by the patient's blood pressure.

3. Third-line therapy is reserved for patients who are resistant to first- and second-line agents and for those who develop specific complications.

a. Nesiritide

(1) Multiple physiologic effects

(a) Hemodynamic (vasodilator, reduces preload and afterload)

(b) Diuretic (promotes free water and sodium loss)

(c) Neurohormonal (vasodilator, reduces aldosterone levels, inhibits renin-angiotensin system)

(2) Clinical trials do not clearly demonstrate efficacy of nesiritide as a third-line agent. In addition, studies have demonstrated an increased mortality rate and impaired renal function after a single infusion; therefore, its use is not recommended.

b. Intra-aortic balloon counter pulsation

(1) Indicated for cardiogenic shock refractory to inotropes

(2) Works by "unloading" the heart during systole, as well as by increasing coronary and cerebral blood flow during diastole

(3) Can be a life-saving intervention for patient stabilization before thrombolytic therapy or angioplasty

c. Norepinephrine

(1) The pressor agent of choice for those in profound cardiogenic shock unresponsive to inotropic therapy; if not administered before this time (ie, as second-line therapy) for hypotension, consider it now.

(2) Its use should be viewed as a necessary temporary measure to maintain coronary perfusion while other rescue strategies (angioplasty, balloon pumping, surgery) are being arranged.

(3) Concomitant use of additional agents should be considered.

(a) Dopamine: in lower doses, may blunt renal vasoconstriction → ↑ renal perfusion → ↑ urine output

(b) Dobutamine: once blood pressure is restored, may ↑ cardiac output

(4) Dosage: start at 1–4 mcg/min IV, and titrate upward as needed.

B Management of heart failure without pulmonary congestion

1. Patients present with hypotension, jugular venous distention, and clear lung fields.
2. This presentation is common in patients with inferior wall MIs associated with right ventricular infarction, which is present in one-third of inferior wall MIs.
3. Hypotension in these patients is due to a decrease in left ventricular preload and should be treated with small boluses (250 mL) of an isotonic crystalloid solution (up to 2 L). Multiple fluid boluses as well as inotropic support may be needed to provide sufficient preload and correct the blood pressure.
4. Nitrates and diuretics can significantly and rapidly decrease blood pressure and should be used with caution (if at all) in these patients.

CARDIOMYOPATHIES AND SPECIFIC HEART MUSCLE DISEASES

I. CARDIOMYOPATHIES

A. Definition

1. Diseases of the heart muscle of unknown etiology that produce both structural and functional damage to the myocardium
2. Classified as dilated (most common), restrictive, or hypertrophic based on differences in pathophysiology and clinical presentation (however, there is some overlap).

B. Idiopathic dilated (congestive) cardiomyopathy

1. Characterized by dilatation of all four chambers (ventricles > atria), increased myocardial mass (hypertrophy), and systolic pump failure. There is a high association of idiopathic dilated cardiomyopathy with viral myocarditis; unexplained heart failure is commonly the only manifestation.
2. Clinical presentation
   a. History
      (1) Signs and symptoms of left- and right-ventricular CHF (dyspnea on exertion and fatigue, which are the two most common complaints), paroxysmal nocturnal dyspnea, orthopnea, palpitations, dependent edema, ascites) and/or
      (2) Manifestations of systemic or peripheral embolization (acute neurologic deficit, flank pain and hematuria, pulseless cyanotic extremity)
   b. Physical findings
      (1) Bibasilar rales
      (2) Peripheral edema, jugular venous distention, and hepatomegaly (when associated with a pulsatile liver, tricuspid regurgitation is also present)
      (3) Abnormal heart sounds
         (a) S3 and S4 gallop (most common auscultatory findings)
         (b) High-pitched systolic ejection murmur of mitral regurgitation (best heard at apex)
         (c) Holosystolic ejection murmur of tricuspid regurgitation (best heard at lower left sternal border)
         (d) Tachycardia (often with a narrow pulse pressure) when CHF is present
3. Diagnostic evaluation
   a. ECG
      (1) Left ventricular or biventricular hypertrophy
      (2) Left atrial or biatrial enlargement
      (3) Poor R-wave progression
      (4) A new bundle-branch block (may occur before any signs of chamber dilation or pump dysfunction)
      (5) AV block (usually first- or second-degree)
      (6) Atrial fibrillation (most common dysrhythmia)

b. Chest radiograph
   (1) Cardiomegaly with enlargement of all four chambers
   (2) Pulmonary venous congestion
c. Echocardiography
   (1) Enlargement of ventricles and atria
   (2) Increased systolic and diastolic volumes
   (3) Decreased ejection fraction (<45%)
   (4) Abnormal ventricular contractility (the sine qua non of idiopathic dilated cardiomyopathy)
   (5) Mitral and tricuspid valve regurgitation
   (6) Mural thrombi

4. Management
   a. Aimed at alleviating symptoms and generally includes a regimen of diuretics, digitalis, and vasodilators (nitroprusside, nitrates, prazocin, hydralazine, ACE inhibitors)
   b. Anticoagulants should be administered to patients with:
      (1) Intracardiac thrombi
      (2) Evidence of pulmonary or systemic thromboembolism
      (3) Chronic atrial fibrillation

C. Restrictive cardiomyopathy

1. Characterized by diastolic restriction of ventricular filling; the end-diastolic ventricular volume is low, the end-diastolic ventricular pressure is high, and cardiac output is decreased—a hemodynamic picture that mimics that of constrictive pericarditis.
2. Clinical presentation
   a. History
      (1) Similar to that of patients with dilated cardiomyopathy
      (2) Signs and symptoms of systemic and pulmonary venous congestion; however, evidence of right-sided CHF often predominates.
      (3) Exercise intolerance is a common complaint.
   b. Physical examination (similar to that of idiopathic dilated cardiomyopathy)
      (1) Bibasilar rales
      (2) Peripheral edema, jugular venous distention, hepatomegaly
      (3) Abnormal heart sounds
         (a) S_3 and S_4 gallop (common)
         (b) Systolic ejection murmur of mitral or tricuspid regurgitation
         (c) Tachycardia
      (4) The apex impulse is usually easily palpable and mitral regurgitation is more common (in contrast to constrictive pericarditis). Other signs that are not present in constrictive pericarditis are a gallop rhythm and a positive Kussmaul sign (jugular venous distention on inspiration).
3. Diagnostic evaluation
   a. ECG
      (1) Chamber enlargement (atria > ventricles)
      (2) Nonspecific ST–T-wave changes

(3) Low voltage
(4) Dysrhythmias (especially atrial fibrillation) are common.

b. Chest radiograph
(1) May be normal initially (a small heart suggests constrictive pericarditis)
(2) Cardiomegaly with pulmonary venous congestion is seen with disease progression.

c. Echocardiography
(1) Thickened walls
(2) Normal or slightly enlarged ventricular cavity and moderate to markedly dilated atria
(3) Normal or slightly decreased systolic function
(4) Mitral and tricuspid regurgitation

d. If differentiation from constrictive pericarditis is still uncertain after echocardiography, then CT, MRI, or combined Doppler and two-dimensional echocardiography may be required to make the diagnosis → thickened pericardium in constrictive pericarditis. Biopsy is the gold standard to exclude constrictive pericarditis if all else fails.

4. Management
a. Aimed at alleviating symptoms and usually includes a regimen of diuretics and digitalis
b. In the absence of left ventricular enlargement, administration of digitalis may be without beneficial effect.
c. Vasodilators reduce afterload but produce hypotension and have not been found to be beneficial.

D. Hypertrophic cardiomyopathy

1. Characterized by left ventricular hypertrophy without associated ventricular dilatation. The hypertrophy is generally asymmetric, involving the septum to a greater extent than the free wall; on histologic examination, the myocardial fibers have a marked and extensive disorganized whorled pattern (particularly in the septum). Other common features include ventricular cavities that are reduced in size, atrial dilatation, mitral valve thickening, impaired diastolic relaxation, and restricted left ventricular filling.
2. In >50% of cases, this disorder is inherited via an autosomal dominant transmission pattern with variable penetration. The remaining cases appear to be sporadic.
3. Clinical presentation
a. History
(1) Dyspnea on exertion (most common initial complaint)
(2) Atypical chest pain (poor response to nitroglycerin is common); chest pain that is relieved by assuming the recumbent position is pathognomonic of hypertrophic cardiomyopathy but rarely encountered.
(3) Palpitations
(a) Ventricular and atrial dysrhythmias are common.
(b) Tachydysrhythmias (particularly atrial fibrillation) are poorly tolerated and may require emergent intervention.
(4) Syncope and presyncope
(a) Usually related to exertion
(b) Due to dysrhythmias or a sudden decrease in cardiac output
(5) Sudden death (usually due to dysrhythmias that occur with exercise, especially ventricular tachycardia)

(6) Symptom severity correlates with the degree of hypertrophy which, in turn, correlates with the patient's age, ie, the older the patient, the more severe the symptoms.

b. Physical examination

(1) A prominent "a wave" may be noted on inspection of the neck veins, ie, a pulse wave that reflects the powerful systolic pressure of the hypertrophied left ventricle; this should not be confused with jugular venous distention, because jugular venous pressure is not usually increased.

(2) Rapid biphasic carotid pulse (pulsus biferiens)

(3) Abnormal heart sounds

(a) S_4 gallop: prominent systolic ejection murmur along the lower left sternal border and at the apex with radiation to the axilla

(b) This murmur is the result of left ventricular outflow obstruction and mitral regurgitation; it is increased with maneuvers or therapies that decrease left ventricular end-diastolic volume: the Valsalva maneuver, sudden standing, exercise, amyl nitrate inhalation, and administration of isoproterenol. Remember to perform one of these maneuvers when evaluating young patients presenting with exertional syncope or chest pain.

4. Diagnostic evaluation

a. ECG: almost always abnormal

(1) Left ventricular hypertrophy and left atrial enlargement

(2) Changes in the anterior, lateral, or inferior leads

(a) "Septal Q waves" >0.3 mV (represent septal depolarization)

(b) "Dagger-like" deep, narrow (<0.04 sec) lateral Q waves

(c) Upright T waves in leads with qR or qS complexes (represent mid-ventricular obstruction)

(d) Giant inverted T waves in lateral leads (represent obstruction that is primarily localized in ventricular apex)

(e) The diagnosis of hypertrophic cardiomyopathy should be considered in any young patient whose ECG suggests MI but who does not have a history of infarction.

(3) Paroxysmal or sustained atrial fibrillation and premature ventricular contractions are the most common dysrhythmias.

b. Chest radiograph: usually normal

c. Echocardiography

(1) Left ventricular hypertrophy with disproportionate septal hypertrophy

(2) Small left ventricular cavity

(3) Systolic anterior motion of the mitral valve (a highly specific finding but not very sensitive)

5. Management

a. Patients with hypertrophic cardiomyopathy who present with unstable atrial fibrillation (severe CHF, cardiogenic shock) require immediate cardioversion and heparinization (preferably with a low-molecular-weight heparin such as enoxaparin 1 mg/kg SC). Risk of sudden death is high, not only from hemodynamic compromise but also from thromboembolism. Rate control and anticoagulation are of paramount importance in this situation. Atrial fibrillation can cause dramatic hemodynamic compromise,

because hypertrophic cardiomyopathy patients have restricted left ventricular filling and, therefore, depend on atrial contraction to maintain efficient flow; when atrial fibrillation occurs, cardiac output drops suddenly.

b. ß-blockers (eg, propranolol)
   (1) Improve most symptoms of this disease (dyspnea, chest pain, dizziness, syncope) and so are the mainstay of therapy
   (2) Decrease myocardial oxygen consumption by decreasing exertion-related outflow obstruction
c. Calcium channel blockers (eg, verapamil, nifedipine)
   (1) May be useful in select patients who do not respond to ß-blockers
   (2) Decrease outflow obstruction, myocardial oxygen consumption, and incidence of angina; increase exercise capacity
   (3) Avoid in patients with conduction blocks or increased pulmonary venous pressures (CHF).
d. Amiodarone
   (1) The treatment of choice for ventricular dysrhythmias; also indicated for patients who do not respond to ß-blockers and calcium channel blockers.
   (2) Controls most atrial and ventricular dysrhythmias and appears to prevent sudden cardiac death (the most common cause of death in these patients)
e. Diuretics are useful in the setting of pulmonary and venous congestion but must be used with caution to avoid hypotension.
f. Agents that either increase myocardial contractility (digitalis, ß-adrenergic agents) and/or reduce ventricular volume (eg, nitrates, other vasodilators) should be avoided, because they can increase outflow obstruction.
g. Surgical management (most common procedure is septal myomectomy)
   (1) Reserved for very symptomatic patients with large systolic gradients (>50 mmHg) who do not respond to drug therapy
   (2) Effectively relieves left ventricular outflow obstruction in 95% of patients but has a mortality rate of 3%–8%
h. Antibiotic prophylaxis is no longer indicated for dental or routine emergency department procedures.
i. Avoiding competitive athletics is advised, because sudden death can follow vigorous exertion.

II. SPECIFIC HEART MUSCLE DISEASES

A. Definition

1. Disorders of the heart muscle associated with a single known cause or systemic disease
2. These diseases were previously referred to as secondary cardiomyopathies, but this terminology is no longer used.

B. Categories

1. Toxins
   a. Most commonly associated with dilated cardiomyopathy

(1) Ethanol
(2) Cobalt, cocaine
(3) Lithium
(4) Doxorubicin (adriamycin)
(5) Daunorubicin
(6) Emetine

b. Others
(1) Heavy metals
(2) Amphetamines

2. Nutritional deficiencies
a. Thiamine (B_1) deficiency (beriberi)
b. Vitamin C deficiency (scurvy)
c. Vitamin B_6 deficiency (pellagra)
d. Selenium deficiency (Keshan disease)
e. Kwashiorkor

3. Metabolic
a. Most commonly associated with restrictive cardiomyopathy
(1) Hemochromatosis
(2) Glycogen storage disease Type II

b. Others
(1) Hypothyroidism (myxedema)
(2) Hyperthyroidism (thyrotoxicosis)
(3) Uremia
(4) Pheochromocytoma
(5) Hypophosphatemia

4. Infiltrative (most commonly associated with restrictive cardiomyopathy)
a. Amyloidosis (most common cause in the Western hemisphere)
b. Sarcoidosis
c. Endomyocardial fibrosis (most common cause worldwide)

5. Collagen vascular diseases

6. Neuromuscular
a. Muscular dystrophies
b. Friedreich ataxia
c. Myasthenia gravis

7. Myocarditis
a. Viral
b. Bacterial
c. Parasitic (Chagas' disease due to *Trypanosoma cruzi*)
d. HIV-associated

8. Peripartum (associated with dilated cardiomyopathy)

9. Ischemia

10. Radiation

PERICARDIAL DISORDERS

I. PERICARDITIS

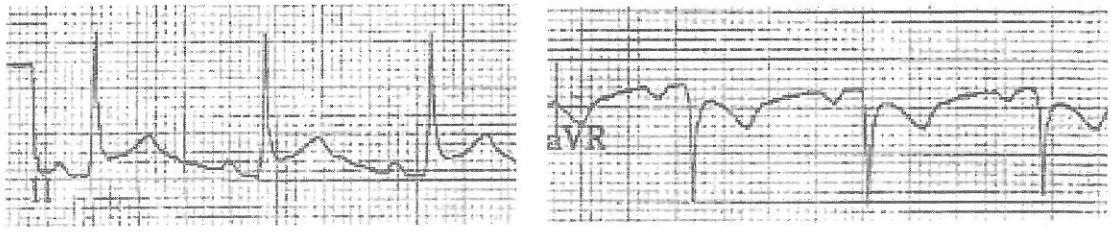
A. Etiology
1. Idiopathic (along with viral, most common causes)
2. Infectious agents
   a. Viral (along with idiopathic, most common causes)
      (1) Coxsackie viruses A and B
      (2) Echovirus
      (3) Adenovirus
      (4) HIV
      (5) Epstein-Barr virus
      (6) Influenza
      (7) Hepatitis B
   b. Bacterial (uncommon cause)
      (1) *Staphylococcus*
      (2) *Pneumococcus*
      (3) *Streptococcus*
      (4) *Meningococcus*
      (5) *Mycobacterium* sp
      (6) *Rickettsia* sp
      (7) *Borrelia burgdorferi*
      (8) *Mycoplasma*
   c. Fungal
      (1) Histoplasmosis
      (2) Blastomycosis
      (3) Coccidiomycosis
3. Malignancies
   a. Metastatic
      (1) Breast
      (2) Lung
      (3) Melanoma
      (4) Leukemia
      (5) Lymphoma
   b. Primary pericardial tumors (mesotheliomas): rare cause
4. Systemic illnesses
   a. Systemic lupus erythematosus
   b. Acute rheumatic fever
   c. Rheumatoid arthritis
   d. Scleroderma

e. Polyarteritis nodosa
f. Sarcoidosis
g. Myxedema
h. Amyloidosis

5. Medications
   a. Anticoagulants
   b. Procainamide
   c. Hydralazine
   d. Isoniazid
6. Radiation
7. Cardiac injury
   a. Acute MI
   b. Dressler syndrome (late post-MI pericarditis)

B. Diagnosis

1. Clinical presentation
   a. History
      (1) Sharp precordial or retrosternal chest pain exacerbated by inspiration, swallowing, or movement of the upper torso (pain is increased in the supine position but relieved by sitting up and leaning forward); radiation to the trapezius muscle ridge (especially the left) is common and is a distinctive characteristic.
      (2) Dyspnea
      (3) Low-grade, intermittent fever
   b. Physical examination: pericardial friction rub is pathognomonic.
      (1) May be positional and intermittent
      (2) Best appreciated when the patient is sitting and leaning forward or is in the hands-and-knees position
      (3) Scratchy in character; best heard with the diaphragm of the stethoscope positioned along the lower left sternal border or cardiac apex
      (4) Classically triphasic (with presystolic, systolic, and diastolic components) but may be biphasic or monophasic
2. ECG
   a. Normal sinus rhythm or sinus tachycardia; dysrhythmias are uncommon.
   b. Initial ST and PR segment changes
      (1) Diffuse, nonanatomic ST-segment elevation with upward concavity is seen acutely in multiple leads except aVR and V_1. ST-segment elevation due to acute MI may also be concave upward but can also be flat or convex in configuration and has a nondiffuse, anatomic distribution (eg, inferior or anterior wall pattern); ST elevation in III > II → think MI
      (2) ST-segment depression in leads aVR and V_1 is common but nonspecific; however, ST-segment depression in any of the other leads strongly suggests MI and not pericarditis.
      (3) PR-segment depression in multiple leads is common with viral pericarditis, most prominent in lead II, and often the earliest ECG manifestation.



Courtesy of Dr. Daniel Schwerin

c. **ST and PR segment changes then normalize**
d. **Diffuse T-wave inversion a late sign**
e. **Finally after several weeks, all ECG segments and waves should have returned to baseline.**

3. Chest radiograph: usually normal but, in the presence of a large pericardial effusion (>200 mL), may reveal an enlarged cardiac silhouette
4. Echocardiography: the most sensitive and most specific procedure for detecting the presence and size of an associated pericardial effusion; considered the diagnostic method of choice. Two-dimensional echocardiography can detect as little as 15 mL of fluid and has the advantage of being able to provide information about cardiac function. Transesophageal echocardiography and chest CT are superior to transthoracic echocardiography for demonstration of pericardial thickening (seen in constrictive pericarditis).
5. CT scan
   a. Can define the presence and extent of pericardial effusion but, unlike echocardiography, cannot assess cardiac function and may miss hemopericardium because of similar densities of blood and myocardium
   b. Usually reserved for times when echocardiography is unavailable or yields equivocal results
6. Laboratory studies
   a. CBC: increased WBC count with a left shift is common.
   b. Erythrocyte sedimentation rate: usually increased
   c. BUN/creatinine: useful in excluding uremia
   d. Cardiac enzymes: often minimally increased
   e. Other specific tests (blood cultures, viral titers, thyroid function tests, etc): should be obtained as appropriate, depending on suspected underlying etiology

C. Management

1. **Because they tend to follow a benign, self-limited course, patients with viral or idiopathic pericarditis may be treated as outpatients if reliable follow-up can be assured. All others, particularly those with severe intractable pain or an underlying precipitant that requires specific treatment (MI, uremia, bacterial infection, etc) should be hospitalized.**
2. **Pain relief**
   a. **Anti-inflammatory agents generally provide symptomatic relief within 24 hours of initiation. Use one of the following:**
      (1) Aspirin 650 mg orally every 6 hours
      (2) Ibuprofen 600–800 mg orally every 6–8 hours
      (3) Indomethacin 25–75 mg orally every 6–8 hours

(4) Colchicine decreases the duration of symptoms and recurrences when administered during the first episode. The recommended dosage is 2 mg/day for 1–2 days, followed by a maintenance dosage of 1 mg/day (0.5 mg twice per day). The major adverse effect is diarrhea; if this develops, a lower dosage should be used. Additionally, reduced dosages should be used in patients >70 years old, and care should be exercised in the presence of renal insufficiency or if the patient is taking other drugs that are metabolized through the cytochrome P450 system.

(5) Prednisone (60 mg/day orally) should not be administered in the acute setting for patients with acute pericarditis. Steroids have been found to be an independent risk factor for recurrences.

b. Opiate medications may be used when immediate pain relief is needed.

3. Treat the underlying disorder.
   a. Antimicrobial agents for patients with underlying infections
   b. Dialysis for uremic patients
   c. Cessation of the causative agent for medication-induced pericarditis
4. Pericardiocentesis for tamponade

II. PERICARDIAL TAMPONADE

A. Classic clinical presentation

1. Beck triad (late findings occurring just before cardiac arrest)
   a. Hypotension
   b. Jugular venous distention
   c. Distant or "muffled" heart tones (quiet heart)
2. Narrow pulse pressure
3. Dyspnea
4. Tachycardia (earliest sign)
5. Pulsus paradoxus >10 mmHg
6. Kussmaul sign: a rise in central venous pressure (by observation of jugular venous pulsation) with spontaneous inspiration

B. Diagnostic evaluation

1. ECG
   a. Low QRS voltage: a nonspecific finding of pericardial effusion
   b. Total electrical alternans: a beat-to-beat alternating pattern primarily affecting the QRS complex that occurs from shifting pericardial fluid and heart position—highly suggestive of the presence of large pericardial effusions, but this finding is poorly sensitive
2. Chest radiograph: in patients with chronic effusions, may reveal an enlarged "water bottle"–shaped cardiac silhouette without associated pulmonary redistribution; this is not the case in acute traumatic tamponade, in which the cardiac silhouette is typically normal.

3. Echocardiography
   a. The gold standard for diagnosing pericardial effusion; it can quickly and accurately confirm the presence of tamponade.
   b. If rapidly available and the patient's condition allows, it should be performed whenever pericardial tamponade is suspected.
   c. Findings consistent with tamponade
      (1) A large pericardial effusion, visualized as an echo-free space behind the left ventricle and in front of the right ventricle
      (2) Diastolic collapse of the right ventricle and the right atrium
      (3) Swinging motion of the heart in the effusion (producing electrical alternans)
4. Differential diagnosis in patients with neck vein distention and hypotension
   a. Acute pericardial tamponade
   b. Tension pneumothorax
   c. Massive pulmonary embolism
   d. Acute left ventricular MI with cardiogenic shock
   e. Acute right ventricular MI
   f. Superior vena cava syndrome
   g. Acute valvular dysfunction with regurgitation
5. Clinical picture of a patient who needs immediate pericardiocentesis
   a. Air-hunger and drowsiness or confusion
   b. Thready pulse and pulsus paradoxus that is >50% of the pulse pressure
   c. ↑ jugular venous distention and ↓ systolic blood pressure

C. Management

1. Perform stabilizing/temporizing measures while arranging for pericardiocentesis.
   a. Establish two large-bore IV lines, provide supplemental oxygen (5–10 L/min), and place cardiac monitor.
   b. Provide aggressive volume resuscitation with crystalloid solution or blood.
   c. Administer dobutamine or dopamine as needed for inotropic support.
   d. Obtain immediate cardiology and cardiothoracic surgery consult, depending on availability. Urgent pericardiocentesis can usually be performed in the catheterization laboratory by cardiology, but if the cardiothoracic surgeon is unable to perform the procedure or it is unsuccessful, he or she will likely need to perform pericardiotomy in the operating room.
2. Definitive therapy is pericardiocentesis. Although pericardiocentesis can be performed blindly or by using ECG guidance, ultrasound guidance is safer and is the technique of choice if it can be performed in a timely manner.

MYOCARDITIS

I. ETIOLOGY

A. Viruses: most common cause of myocarditis
   1. Coxsackie A and B (historically most common viral cause)
   2. Adenovirus (increase in incidence in the 1990s)
   3. Parvovirus B19 (increase in incidence in 2000s)
   4. Epstein-Barr
   5. Hepatitis B and C
   6. Human herpesvirus-6
   7. Echovirus
   8. Poliovirus
   9. HIV
   10. Cytomegalovirus
   11. Influenza and parainfluenza

B. Bacteria
   1. ß-hemolytic streptococci (rheumatic fever)
   2. *Corynebacterium diphtheriae*
   3. *Neisseria meningitidis*
   4. *Borrelia burgdorferi* (Lyme disease)
   5. *Mycoplasma pneumoniae*

C. Parasites
   1. Chagas' disease
   2. Toxoplasmosis
   3. Trichinosis

D. Systemic diseases
   1. Kawasaki syndrome
   2. Systemic lupus erythematosus
   3. Sarcoidosis
   4. Giant-cell myocarditis

E. Drug hypersensitivity
   1. Sulfonamides
   2. Penicillins
   3. Methyldopa

F. Toxins
   1. Cocaine
   2. Inhalants (eg, toluene)

II. CLINICAL PRESENTATION

A. Determined by degree of cardiac involvement; ranges from nonspecific symptoms of fatigue and dyspnea to florid CHF, significant dysrhythmias, and sudden death.

B. History of a preceding or concurrent viral illness is common.

C. Often presents in association with acute pericarditis

D. Signs and symptoms
   1. Fever and retrosternal or precordial chest pain (frequent complaints)
   2. Fatigue, palpitations, dizziness, and/or syncope
   3. Signs and symptoms of CHF (dyspnea, rales, peripheral edema, jugular venous distention)
   4. Sinus tachycardia disproportionate to the degree of fever (>101°F [38.5°C]) present and probably secondary to associated heart failure
   5. Cardiac dysrhythmias or conduction disturbances
   6. Abnormal heart sounds
      a. Pericardial friction rub (when associated pericarditis is present)
      b. Soft S_1
      c. S_3 or S_4 gallop
      d. Murmurs of mitral or tricuspid regurgitation

E. **Suspect giant-cell myocarditis in patients with ventricular tachycardia, heart block, and progressively worsening clinical course despite therapy; high risk of death and often require cardiac transplant**

III. DIAGNOSTIC EVALUATION

A. ECG
   1. Nonspecific ST–T wave changes (may be localized or diffuse)
   2. Dysrhythmias ranging from sinus tachycardia (most common) to atrial or ventricular dysrhythmias
   3. Conduction disturbances (AV block, bundle-branch block)
   4. Low QRS voltage
   5. Pseudoinfarction patterns, nonspecific ST–T wave changes

B. Chest radiograph
   1. Generally normal
   2. Cardiomegaly, pulmonary venous hypertension, and/or pulmonary edema may be present.

C. Echocardiography
   1. Demonstrates dilated chambers with either diffuse hypokinesis or focal wall motion abnormalities
   2. Most useful to exclude other causes of heart failure

D. Laboratory studies

1. CBC reveals mild to moderate leukocytosis.
2. Erythrocyte sedimentation rate is increased.
3. Cardiac enzymes
   a. Characteristically rise and fall slowly over a period of days (unlike the rapid rise seen in acute MI)
   b. Troponin I levels are more sensitive and specific than creatine kinase-MB levels.

E. Endomyocardial biopsy

1. Can provide definitive diagnosis but suffers from poor sensitivity and specificity
2. Most indicated for evaluation of suspected giant-cell myocarditis or in cases of fulminant myocarditis

IV. MANAGEMENT

A. Primarily supportive

1. Admit to ICU.
2. Bed rest; avoid strenuous activity.

B. Pharmacotherapy

1. Administer antibiotics if an underlying bacterial cause is present.
2. Immunosuppressive agents (steroids, cyclosporin) and NSAIDs are contraindicated in early myocarditis.
3. High-dose IV gamma globulin beneficial in preliminary studies in children with Kawasaki syndrome.
4. Antiviral agents (eg, interferon) are currently under investigation.
5. Manage CHF with the usual drug protocols; however:
   a. Digoxin should be used with caution, because the inflamed myocardium is very sensitive to it.
   b. ACE inhibitors (eg, captopril) are particularly beneficial in that they decrease cellular necrosis and inflammation.
6. Manage dysrhythmias with the standard antidysrhythmic agents and electrical therapies.
   a. Amiodarone recommended for ventricular dysrhythmias.
   b. Patients may require automatic implantable cardioverter defibrillator for persistent ventricular dysrhythmias.
   c. Temporary or permanent implanted pacemaker may be needed for persistent heart block.

VALVULAR HEART DISEASE

I. MITRAL VALVE PROLAPSE ("CLICK MURMUR SYNDROME")

A. Epidemiology and etiology

1. Most common valvular heart disease; present in 5%–10% of the population, with young women most frequently affected (female-to-male ratio of 2:1).
2. It can occur as an autosomal dominant congenital disorder or as part of a connective tissue disorder (Ehlers-Danlos syndrome, Marfan syndrome) in association with skeletal abnormalities (such as severe scoliosis, "straight" back, pectus excavatum), or it may occur sporadically.

B. Clinical presentations

1. Typical patients
   a. Young woman with palpitations
   b. Young female athlete passed out during a training or practice session. She is anxious and complains of chest pain and palpitations. A tachydysrhythmia and orthostatic hypotension are found on physical examination.
   c. Older man with a syncopal episode at home
2. Physical examination findings
   a. The murmur is a high-pitched, late systolic regurgitant murmur heard best at the apex using the diaphragm of the stethoscope. One or more mid- or late-systolic clicks may also be heard.
   b. Maneuvers or therapies that decrease left ventricular volume (Valsalva maneuver, sudden standing, inhalation of amyl nitrate, isoproterenol infusion) → earlier and greater prolapse → movement of the click closer to S1 and an increase in the duration of the murmur
   c. Maneuvers or therapies that increase left ventricular volume (passive leg raising, maximal isometric handgrip, squatting, phenylephrine infusion) → delay of prolapse until late in systole → movement of the click and murmur away from S1 and a decrease in the duration of the murmur

C. Diagnostic evaluation

1. ECG: although most often normal, findings may include flattened or inverted T waves in the inferior leads (II, III, and aVF) and prolongation of the QT interval.
2. Chest radiograph: unless significant mitral regurgitation is present, the lungs and cardiac silhouette are usually normal.
3. Echocardiography: confirms the diagnosis; two-dimensional echocardiography is more sensitive than M-mode.

D. Complications: although most patients are asymptomatic and without complications, patients may present with any of the following:

1. Atypical (nonexertional, sharp) chest pain: thought to be due to localized ischemia from tension on the papillary muscles or to coronary artery spasm

2. Palpitations, light-headedness, or syncope: due to dysrhythmias (premature atrial contractions, premature ventricular contractions, paroxysmal supraventricular tachycardia, ventricular tachycardia)
3. Sudden death (rare): due to ventricular tachycardia or ventricular fibrillation
4. Transient ischemic attacks or strokes: believed to result from embolization of leaflet thrombi
5. CHF: due to severe mitral valve regurgitation associated with mitral valve prolapse

E. Treatment: reserved for symptomatic patients

1. ß-blockers for patients with chest pain and dysrhythmias
2. Antiplatelet (eg, ASA) or anticoagulant therapy for patients with a history of systemic embolization (transient ischemic attack or stroke)

II. MITRAL STENOSIS

A. Etiology

1. Rheumatic heart disease is responsible for ≥90% of cases of isolated mitral stenosis.
2. Congenital malformations
3. Calcification of the mitral annulus and leaflets
4. Left atrial myxoma

B. Clinical presentation

1. History
   a. Exertional dyspnea (most common symptom)
   b. Hemoptysis (second most common symptom)
   c. Orthopnea and paroxysmal nocturnal dyspnea
   d. Palpitations (premature atrial contractions, paroxysmal atrial fibrillation)
   e. Fatigue
   f. Systemic emboli
2. Physical examination findings
   a. Mitral facies (malar rash)
   b. Palpable diastolic thrill at the apex
   c. Loud S_1
   d. Early diastolic opening snap followed by a low-pitched, rumbling diastolic murmur heard best at the apex

C. Diagnostic evaluation

1. ECG
   a. Left atrial enlargement (P mitrale)
   b. Atrial fibrillation
   c. Right ventricular hypertrophy (if marked pulmonary hypertension is present)
2. Chest radiograph
   a. Straightening of the left heart border due to left atrial enlargement
   b. Calcification of the mitral annulus and leaflets

c. Findings of pulmonary congestion
d. Right ventricular lift (or hypertrophy) if pulmonary hypertension is present

D. Complications

1. Atrial fibrillation (most common)
2. Embolic events, especially with atrial fibrillation
3. Frequent respiratory infections
4. Infective endocarditis (rare)
5. Massive pulmonary hemorrhage from rupture of pulmonary bronchial venous connections

E. Management

1. Rate control with IV diltiazem or digoxin for atrial fibrillation with rapid ventricular response
2. Blood transfusion and, possibly, surgery for massive hemoptysis
3. Diuretics for pulmonary congestion
4. Anticoagulation for patients with systemic embolization and atrial fibrillation

III. MITRAL REGURGITATION

A. Can be acute or chronic; these two disease entities differ significantly in terms of their causation, disease course, presentation, and management.

B. Acute mitral regurgitation is the result of chordae tendineae, papillary muscle, or valve leaflet rupture and is abrupt in onset.

1. Etiology
   a. Acute MI
   b. Infectious endocarditis
   c. Trauma
2. Clinical presentation
   a. Symptoms are those of fulminant CHF/pulmonary edema (eg, dyspnea) and those of the disease process that precipitated the event (eg, chest pain in the acute MI patient)
   b. Physical examination findings
      (1) Tachycardia, tachypnea, and rales
      (2) Hypotension
      (3) Jugular venous distention with a prominent cannon "a" wave
      (4) Palpable thrill at apex
      (5) Soft S_1
      (6) Loud apical systolic murmur (radiating to the axilla) that can be holosystolic, late systolic, or crescendo-decrescendo
      (7) S_3 and S_4 (a common finding)

3. Diagnostic evaluation
   a. ECG
      (1) Absence of left atrial enlargement and left ventricular hypertrophy
      (2) Sinus tachycardia
   b. Chest radiograph
      (1) Normal cardiac silhouette
      (2) Severe pulmonary edema
4. Management
   a. Airway and hemodynamic support (may include intraaortic balloon pump to augment cardiac output)
   b. Supplemental oxygen
   c. Afterload reduction
   d. Emergent consult with a cardiothoracic surgeon
   e. Treatment of underlying cause (eg, antibiotics for infectious endocarditis)

C. Chronic mitral regurgitation evolves slowly and frequently coexists with mitral stenosis.

1. Etiology
   a. Rheumatic heart disease is most common cause.
   b. Mitral valve prolapse
   c. Connective tissue disorders (eg, Marfan syndrome)
2. Clinical presentation
   a. History
      (1) Exertional fatigue or dyspnea
      (2) Orthopnea and paroxysmal nocturnal dyspnea
      (3) Systemic emboli
   b. Physical examination findings
      (1) Left parasternal heave and thrill
      (2) High-pitched holosystolic murmur that radiates to the axilla
      (3) Soft S_1 that is often obscured by the murmur
      (4) Wide-split S_2
      (5) S_3 and S_4 are common with severe regurgitation.
3. Diagnostic evaluation
   a. ECG
      (1) Left atrial enlargement
      (2) Left ventricular hypertrophy with strain
      (3) Atrial fibrillation (75% of patients)
   b. Chest radiograph
      (1) Left atrial enlargement
      (2) Left ventricular enlargement
      (3) Pulmonary congestion

4. Management
   a. Rate control of atrial fibrillation with diltiazem or digoxin
   b. Diuretics for symptoms of pulmonary congestion
   c. Anticoagulation if atrial fibrillation is present

IV. AORTIC STENOSIS

A. Etiology

1. Congenital bicuspid valve (most common cause in patients <65 years old)
2. Rheumatic heart disease (associated mitral valve disease is common)
3. Calcific aortic stenosis (most common cause in patients >65 years old)

B. Clinical presentation

1. Symptoms appear late in the disease process; patients remain relatively asymptomatic until the valve opening decreases to <1 cm.
   a. Dyspnea on exertion
   b. Angina
   c. Exertional syncope
   d. Symptoms of heart failure
   e. Sudden death
2. Physical examination findings
   a. Carotid pulse is diminished and delayed (pulsus parvus and tardus)
   b. Pulse pressure may be narrowed (<30 mmHg)
   c. Palpable left chest heave
   d. Harsh systolic crescendo-decrescendo murmur heard best at base with radiation into the carotids; also heard on auscultation:
      (1) Absent S_2 component
      (2) Ejection click
      (3) S_4 gallop

C. Diagnostic evaluation

1. ECG
   a. Left ventricular hypertrophy
   b. Left or right bundle-branch block
2. Chest radiograph
   a. Left ventricular enlargement
   b. Poststenotic dilatation of the ascending aorta (a characteristic finding)
   c. Pulmonary vascular congestion (if heart failure is present)
   d. Aortic valve calcification

D. Management

1. Patients with mild symptoms should be advised to avoid strenuous activities.

2. Patients with CHF require hospital admission; administration of medications that decrease preload or afterload in these patients can produce sudden decompensation and should be administered with caution.
3. Symptomatic patients are at high risk of sudden death; they should be referred for valve replacement (definitive therapy) or valvuloplasty (temporizing measure).

V. AORTIC REGURGITATION

A. Can be acute and fulminant or chronic and slowly progressive

B. Acute aortic regurgitation

1. Etiology
   a. Infective endocarditis
   b. Aortic dissection
   c. Trauma
2. Clinical presentation
   a. History
      (1) Severe dyspnea (most common)
      (2) Apprehension
      (3) Other signs of heart failure (eg, paroxysmal nocturnal dyspnea, orthopnea)
      (4) Chest pain
   b. Physical examination findings
      (1) Tachypnea, tachycardia, and inspiratory rales
      (2) Normal or low systolic and diastolic pressures
      (3) Normal pulse pressure (difference of 30–40 mmHg between systolic and diastolic blood pressures)
      (4) Signs of decreased cardiac output (cool, pale extremities, peripheral cyanosis, hypotension, diaphoresis, confusion)
      (5) Diminished or absent S_1
      (6) Loud S_3
      (7) Short diastolic murmur of aortic regurgitation best heard at the left sternal border with the diaphragm of the stethoscope
      (8) Midsystolic flow murmur
3. Diagnostic evaluation
   a. ECG
      (1) Sinus tachycardia
      (2) Nonspecific ST–T wave changes
      (3) Left ventricular strain
   b. Chest radiograph
      (1) Normal cardiac silhouette
      (2) Signs of increased pulmonary venous pressure and pulmonary edema

4. Management
   a. Determine and treat the underlying cause (eg, administer IV antibiotics to patients with bacterial endocarditis).
   b. Treat CHF with supplemental oxygen, diuretics, digitalis, and vasodilators (eg, nitroglycerin, nitroprusside).
   c. Obtain immediate cardiothoracic surgery consult for emergent valve replacement.

C. Chronic aortic regurgitation

1. Etiology
   a. Rheumatic heart disease
   b. Congenital (bicuspid) valve
   c. Prior infective endocarditis
   d. Diseases that dilate the aortic wall (eg, Marfan syndrome, ankylosing spondylitis)
2. Clinical presentation
   a. History
      (1) Exertional fatigue and dyspnea
      (2) Orthopnea, paroxysmal nocturnal dyspnea
      (3) Palpitations
      (4) Angina
      (5) Pulmonary edema
   b. Physical examination findings
      (1) Wide pulse pressure
      (2) Displaced, hyperdynamic point of maximal impulse
      (3) Soft S_1
      (4) High-pitched, decrescendo diastolic blowing murmur best heard along the left sternal border (sine qua non of aortic regurgitation)
      (5) Rapid and forceful carotid upstroke with dramatic collapse (Corrigan or water-hammer pulse)
      (6) Head bobbing with each heartbeat (deMusset sign)
      (7) Prominent nail pulsations (Quincke pulse)
      (8) Singsong murmur over the femoral artery (Duroziez murmur)
      (9) A presystolic or mid-diastolic murmur (Austin-Flint murmur) may be present with severe aortic regurgitation.
3. Diagnostic evaluation
   a. ECG
      (1) Left ventricular hypertrophy
      (2) Conduction abnormalities such as left bundle-branch block
   b. Chest radiograph
      (1) Cardiomegaly
      (2) Aortic root dilatation
      (3) Pulmonary vascular congestion
4. Management: treat CHF with supplemental oxygen, diuretics, vasodilators, and digitalis.

VI. TRICUSPID STENOSIS

A. Etiology
1. Rheumatic heart disease
2. Endocarditis secondary to IV drug abuse

B. Clinical presentation
1. History: patients complain of fatigue and symptoms due to increased systemic venous pressure (eg, edema).
2. Physical examination
   a. Diastolic murmur best heard along the left sternal border and accentuated with inspiration
   b. Peripheral edema
   c. Hepatosplenomegaly
   d. Ascites
   e. Jugular venous distention and giant jugular cannon "a" waves
3. Tricuspid stenosis rarely exists in isolation. Coexisting mitral and aortic valve disease is common and, when present, typically dominates and determines the clinical course.

C. Diagnostic evaluation
1. ECG
   a. Tall, pointed P waves if sinus rhythm is present
   b. Atrial fibrillation
2. Chest radiograph: enlarged right atrium

D. Management
1. Rate control with IV diltiazem for atrial fibrillation with rapid ventricular response
2. Anticoagulation for chronic atrial fibrillation
3. Treatment of underlying cause (eg, IV antibiotics for infectious endocarditis)

VII. TRICUSPID REGURGITATION

A. Etiology
1. Right ventricular dilatation secondary to pulmonary hypertension
2. Rheumatic heart disease
3. Infective endocarditis
4. Trauma

B. Clinical presentation
1. History
   a. Fatigue and dyspnea on exertion
   b. Peripheral edema
   c. Throbbing in the neck and abdomen
   d. Anorexia

2. Physical examination
   a. Palpable left ventricular heave
   b. Prominent systolic pulsations of the jugular veins known as "v" waves or "CV" waves
   c. Holosystolic murmur best heard at the xiphoid area adjacent to the left sternal border

C. Diagnostic evaluation
   1. ECG
      a. Right atrial enlargement
      b. Right ventricular enlargement
      c. Atrial fibrillation (80% of patients)
   2. Chest radiograph
      a. Right atrial enlargement
      b. Right ventricular enlargement
      c. Normal pulmonary vasculature

D. Management: patients in atrial fibrillation require rate control with IV diltiazem and long-term anticoagulation.

VIII. PROSTHETIC VALVES

A. Types and characteristics: there are two types of prosthetic valves (mechanical and bioprosthetic), each with its own particular characteristics.
   1. Mechanical (nontissue) valves
      a. Manmade materials
      b. Life span >20 years
      c. Typically make a loud metallic closure sound and softer opening click and, when located in the aortic position, an associated systolic ejection murmur is normally present.
      d. Require life-long systemic anticoagulation
      e. Cause greater hemolysis and are more thrombogenic than tissue prostheses
      f. Common designs include the tilting disc variety (eg, Bjork-Shiley, Medtronic-Hall), the caged ball variety (eg, Starr-Edwards), and the bileaflet variety (eg, St. Jude).
   2. Bioprosthetic (tissue) valves
      a. Made with human, porcine, or bovine tissue cups
      b. Life span 8–10 years
      c. Make opening and closing sounds that are similar to (but louder than) those of native valves
      d. Except for the initial postoperative period and patients in atrial fibrillation, anticoagulation is optional; ASA is sufficient in most patients.
      e. Cause less hemolysis and are less thrombogenic than mechanical valves

B. Complications and their clinical presentations
   1. Thromboembolic events
      a. Most serious complication of prosthetic valves; occur more frequently with mechanical valves than with bioprosthetic ones

b. Patients with valve dysfunction secondary to thrombus formation typically present with acute onset of CHF, hypotension, and muting or loss of the prosthetic valve sounds. Signs and symptoms in patients with embolic events depend on the location of the emboli and may include paralysis, aphasia, abdominal pain, chest pain, or a cold extremity.

2. Primary valve failure
   a. Significant cause of morbidity and mortality
   b. May result in regurgitant blood flow, acute valvular occlusion, embolization of a prosthetic fragment, or severe hemolysis
   c. The Bjork-Shiley 60° and 70° convexoconcave valves were taken off the market, because they were associated with a high incidence of strut fracture with resultant embolization of the disc. Patients with this complication present with sudden onset of CHF, hypotension, loss of the metallic valve sound, and a new regurgitant murmur. Although these valves are no longer manufactured, many patients still have them.
3. Paravalvular leak
   a. A portion of the prosthetic valve becomes unseated from the valve annulus.
   b. If immediately after surgery, it is usually due to suture disruption, whereas delayed leaks are generally due to endocarditis.
   c. More common with mechanical valves
   d. Patients typically present with sudden onset of pulmonary edema or severe hemolytic anemia. Physical examination reveals a regurgitant murmur.
4. Endocarditis
   a. The causative organism varies with the length of time the valve has been present; <2 months after surgery, *Staphylococcus aureus* and *Streptococcus epidermidis* are the most common organisms. After this time period, non-viridans streptococci are the most frequent organisms.
   b. The diagnosis should be suspected in any patient with a prosthetic valve who presents with fever, particularly if a new regurgitant murmur is present. Laboratory studies typically reveal a leukocytosis with a left shift, anemia (hematocrit <34%), and an increased erythrocyte sedimentation rate.
5. Hemolysis
   a. Common problem with mechanical valves but rarely occurs with tissue valves
   b. Usually low-grade in nature and easily treated with iron and folate supplementation
   c. Presence of severe hemolysis suggests the possibility of a paravalvular leak. These patients typically complain of fatigue, demonstrate orthostatic changes, and/or are jaundiced on examination.
6. Degeneration
   a. Results in the valve becoming incompetent
   b. Presenting symptoms are those of ongoing or accelerating CHF.

C. Evaluation and management of complications

1. Assess and stabilize the ABCs: establish IV of normal saline or lactated Ringer's, provide supplemental oxygen, place patient on cardiac monitor, and check rhythm strip.
2. Determine type, location, and age of the prosthetic valve (most patients carry a card containing this information).
3. Order appropriate diagnostic studies

a. CBC to detect anemia due to hemolysis
b. Lactate dehydrogenase to assess the severity of hemolysis; the degree of increase correlates closely with the degree of hemolysis.
c. Prothrombin time (INR)/partial thromboplastin time to assess adequacy of anticoagulation, especially in patients presenting with thromboembolic events
d. Erythrocyte sedimentation rate, C-reactive protein, and blood cultures, if a fever is present and to evaluate for endocarditis
e. Chest radiograph to assess valve position and look for evidence of vascular congestion
f. Emergency echocardiography or cinefluoroscopy if there is any question of valvular dysfunction

4. Obtain immediate cardiothoracic surgery consult for patients presenting with prosthetic valve dysfunction secondary to thrombus, primary valve failure, paravalvular leak, endocarditis, abscess (perivalvular, intracardiac), or a vegetation that is fungal or large in size.
5. Provide specific therapy for the particular presentation.
   a. Endocarditis IV → antibiotics
   b. Severe anemia → blood transfusion
   c. Thromboembolic event → anticoagulation/thrombolytic therapy
   d. CHF → diuretics

IX. CONDITIONS NECESSITATING ANTIBIOTIC PROPHYLAXIS FOR INFECTIVE ENDOCARDITIS

A. Patients at high risk of infection

1. Prosthetic cardiac valves or prosthetic material used for cardiac valve replacement
2. A history of infective bacterial endocarditis
3. Congenital heart disease (CHD), unrepaired cyanotic CHD, underlying palliative shunts and conduits
4. Completely repaired CHD with prosthetic material or device, whether placed by a surgery or by catheter intervention, during the first 3 postoperative months
5. Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device
6. Valve regurgitation caused by a shunt abnormal valve in a cardiac transplant patient

B. Emergency department procedures that merit prophylaxis

1. Antibiotic prophylaxis is recommended for emergency department patients at high risk (as above) undergoing dental procedures that manipulate the gingiva or periapical region of the tooth or that perforate the oral mucosa.
2. Antibiotic prophylaxis is not recommended for the vast majority of emergency department procedures such as laceration repair, intubation, line placement, or Foley catheterization.
3. No specific recommendations are made for or against prophylaxis for emergency department procedures that involve infected skin (eg, cutaneous abscess drainage) or musculoskeletal tissue, but it is reasonable to administer antibiotics to only high-risk patients.

ENDOCARDITIS

I. DEFINITION

A. A localized infection of the endocardium, the hallmark of which is vegetation

B. Can involve the valve leaflets, the walls of the heart cavities, or the tissue surrounding prosthetic heart valves

II. PATHOPHYSIOLOGY

A. Injury to the endothelium results in formation of a platelet-fibrin complex that is subsequently colonized by microorganisms during periods of transient bacteremia.

B. Although endocarditis does occur in patients with normal valves, patients with congenital/acquired valvular disease and prosthetic valves are most commonly affected.

C. Risk factors

1. Prosthetic valve(s)
2. Congenital valvular heart disease (eg, mitral valve prolapse)
3. Acquired valvular heart disease (eg, rheumatic heart disease)
4. IV drug abuse
5. Calcific valve degeneration
6. Indwelling venous catheters, vascular shunts
7. Hemodialysis
8. Peritoneal dialysis
9. Cardiac surgery
10. HIV infection
11. History of endocarditis
12. Extensive burn injury

D. Causative organisms

1. Although endocarditis is most commonly caused by bacteria, it may also be caused by fungi, rickettsiae, and viruses.
2. The causative organism varies with the type of valve involved (native versus prosthetic) and in the presence of IV drug abuse or immunocompromise.
3. Common bacterial pathogens
   a. Native valves
      (1) Non-*viridans* streptococci (most common)
      (2) *Staphylococcus aureus*
      (3) *Streptococcus viridans*
      (4) Enterococci

b. Prosthetic valves
   (1) Coagulase-negative *Staphylococcus* (early, <60 days after surgery)
   (2) *S aureus* or *Streptococcus epidermidis* (late, > 60 days after surgery)
   (3) *S viridans*
   (4) Enterococci
c. IV drug abusers, immunocompromised
   (1) *S aureus*
   (2) Streptococci
   (3) Gram-negative bacilli

E. Left- versus right-sided disease

1. Left-sided endocarditis → systemic vascular involvement
   a. Most common in patients with acquired valvular disease and congenital heart disease (and is more common than right-sided disease)
   b. Depending on the virulence of the infecting organism, it can present as an acute or subacute illness.
   c. The mitral valve is most commonly affected.
   d. *S viridans* and *S aureus* are the most frequent pathogens.
2. Right-sided endocarditis → pulmonary vascular involvement
   a. Primarily a disease of IV drug abusers
   b. Usually has an acute presentation
   c. Most commonly involves tricuspid valve
   d. Caused by *S aureus* in ~75% of cases

III. CLINICAL PRESENTATION

A. History

1. Protean and nonspecific; may include intermittent fever, chills, sweating, malaise, fatigue, weight loss, chest pain, cough, and neurologic complaints/focal deficits
2. Most common presentation in children is malaise and weight loss.

B. Physical findings

1. Fever (most common finding)
2. Heart murmur
3. CHF
4. Signs of metastatic infection (eg, meningitis, pneumonia)
5. Ophthalmologic signs
   a. Conjunctival hemorrhages
   b. Roth spots (retinal hemorrhages with central clearing)
6. Cutaneous signs
   a. Splinter hemorrhages
   b. Osler nodes (tender erythematous nodules found on the volar surface of the fingertips)

c. Janeway lesions (nontender erythematous macular lesions appearing on the fingers, palms, and soles)
d. Petechiae

7. Neurologic findings
   a. Focal motor deficits
   b. Altered level of consciousness

IV. DIAGNOSTIC EVALUATION

A. Laboratory studies

1. CBC reveals leukocytosis with a left shift and a mild normocytic anemia.
2. Erythrocyte sedimentation rate, C-reactive protein, and serum rheumatoid factor are increased.
3. Urinalysis reveals microscopic hematuria in >50% of patients.

B. Chest radiograph

1. Often unremarkable
2. May reveal septic emboli in patients with right-sided endocarditis

C. ECG

1. Usually normal
2. May demonstrate conductive deficits in the presence of extensive myocardial damage

D. Blood cultures

1. The most useful test for making the diagnosis; positive in >90% of patients with bacterial endocarditis.
2. Three blood cultures from three different sites should be obtained, and each should be evaluated for aerobic, anaerobic, and fungal pathogens.

E. Echocardiography

1. Useful if positive for vegetations, but a negative examination does not exclude the diagnosis.
2. Transesophageal echocardiography
   a. More sensitive in revealing vegetations than transthoracic echocardiography, especially in patients with prosthetic valves.
   b. Recommended as the initial study for low-risk patients
   c. Recommended as the initial study for high-risk patients and as the follow-up study for low-risk patients with a negative or technically inadequate transthoracic echocardiography

V. DIAGNOSIS

A. A presumptive diagnosis of endocarditis should be made in the patient presenting with risk factors and clinical findings (eg, a patient with a prosthetic valve or a recent history of IV drug use and fever).

B. Positive blood cultures and evidence of valvular injury or vegetations usually confirm the diagnosis; if any doubt, a tissue biopsy is the gold standard.

VI. MANAGEMENT

A. Antibiotic therapy

1. Ideally, empiric antibiotic therapy should be started after appropriate blood cultures have been obtained. However, in hemodynamically unstable patients, antibiotic therapy should be started immediately and not delayed while waiting for culture results.
2. The initial antibiotic regimen selected should reflect the susceptibilities of the suspected organism, the acuteness of the presentation, and local resistance patterns.
3. Acceptable empiric antibiotic regimens
   a. In patients with native valves and a subacute presentation:
      (1) Penicillin G (or ampicillin) IV plus nafcillin (or oxacillin) IV plus gentamicin IM or IV *or*
      (2) Vancomycin IV plus gentamicin IM or IV in penicillin-allergic patients
   b. In patients with a prosthetic valve:
      (1) Vancomycin IV plus gentamicin IV plus rifampin orally
      (2) Early consult with a cardiothoracic surgeon should be obtained in all patients with a prosthetic valve.
   c. Vancomycin-resistant enterococcal infections occur, especially in IV drug abusers, patients with prosthetic valves, and those recently hospitalized or taking antimicrobial agents. Current suggested therapy for these patients: quinupristin/ dalfopristin combination plus doxycyline and rifampin.

B. Although ambulatory treatment is possible in certain select cardiovascularly stable patients, patients with endocarditis should generally be admitted to the hospital for treatment, particularly those with prosthetic valves or a history of IV drug abuse.

THORACIC AORTIC DISSECTIONS AND ABDOMINAL AORTIC ANEURYSMS

I. THORACIC AORTIC DISSECTIONS

A. Epidemiology

1. Thoracic aortic dissections are 2–3 times more common than abdominal aortic aneurysm (AAA) ruptures.
2. Men are affected more commonly than women (ratio 3:1).
3. Most patients are 50–70 years old.
4. **Risk factors (>1 increases overall risk)**
   - a. **A history of systemic hypertension: this is the most common predisposing factor, and it is present in 70%–90% of patients.**
   - b. **Connective tissue disorders**
     - (1) **Ehlers-Danlos syndrome**
     - (2) **Marfan syndrome (patients may present when <50 years old)**
     - (3) **Lupus erythematosus (patients may present when <50 years old)**
     - (4) **Giant cell arteritis (patients may present when <50 years old)**
     - (5) **Cystic medial necrosis**
   - c. **Pregnancy (third trimester)**
   - d. **Congenital heart disease**
     - (1) **Bicuspid aortic valve**
     - (2) **Coarctation of the aorta**
   - e. **Turner syndrome**
   - f. **Trauma**
   - g. **Cocaine and methamphetamine use (cause hypertension)**
   - h. **Epstein anomaly**
   - i. **Aortic valve stenosis**
   - j. **Infectious disease (syphilis, endocarditis)**
   - k. **Tobacco use**

B. Pathophysiology

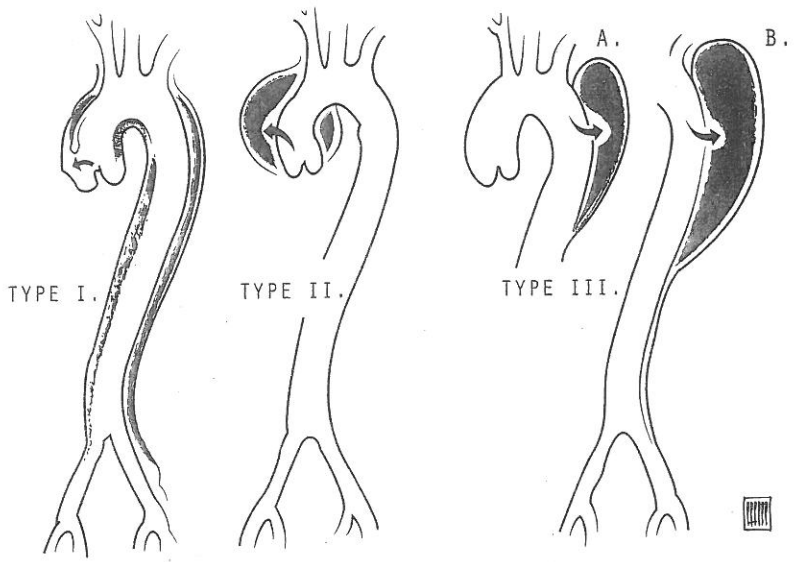
1. Starts with an intimal tear that allows blood to leak into the media and cleave it longitudinally from the adventitia. An important variant of aortic dissection is an intimal tear that arises without classic dissection into the media and produces only a localized bulge in the aortic wall. It is just as deadly as classic dissection and, unfortunately, is not typically seen with the usual tests used to diagnose thoracic aortic dissections.
2. Once the dissection begins, its propagation depends on the blood pressure and the steepness of the pulse wave (dP/dt [rate of change in pressure/time]); high blood pressure and rapid ventricular contractions enhance migration of the dissection.

C. Natural history

1. Most common lethal disease of the aorta
2. Untreated, the mortality rate is:
   a. 33% within 24 hours
   b. 50% within 48 hours
   c. >75% within 2 weeks
   d. 90% within 1–3 months

D. Classification system

1. **Aortic dissections are classified according to the location of the dissecting process. The complications, prognosis, and treatment protocols vary with the location of the dissection. Dissections involving the proximal aorta are more common and more lethal than those confined to the distal aorta. There are two major classification systems: DeBakey and Stanford.**
2. **DeBakey classification**
   a. **Type I: ascending aorta and part of distal aorta (most common type)**
   b. **Type II: ascending aorta only**
   c. **Type III: descending aorta only**
      (1) **Subtype IIIA (extension limited to the diaphragm)**
      (2) **Subtype IIIB (continuation beyond the diaphragm)**



3. **Stanford classification**
   a. **Type A: ascending aorta (DeBakey type I and type II)**
   b. **Type B: descending aorta (DeBakey type III)**

E. Clinical presentation

1. Pain is the most common presenting symptom.
   a. The pain is excruciating, starts abruptly, is maximal from its onset and migrates as the dissection propagates. It generally (but not always) has a tearing or ripping quality and is most commonly located in the chest, upper back, or abdomen.

b. The location of pain provides a clue to the location of the dissection.
   (1) Ascending (proximal) dissections typically present with anterior chest pain (± radiation into the neck, jaw, or arms).
   (2) Arch dissections cause pain in the neck and jaw.
   (3) Interscapular back pain (± radiation into the abdomen or lumbar area) is typical of descending (distal) dissections.

2. Based on the location of the dissection, patients may also present with:
   a. Acute stroke, visual changes
   b. Acute paraplegia, spinal cord deficits
   c. A cool, pulseless extremity
   d. CHF and pulmonary edema
   e. Abdominal pain (mesenteric ischemia), nausea and vomiting
   f. Flank pain, hematuria, or oliguria (impaired renal blood flow)
   g. Syncope
   h. Acute MI (if dissection involves a coronary artery or ostia)
   i. Aortic regurgitation
3. A significant number of patients present with only ischemic or neurologic complications of a "silent" (painless) aortic dissection (most frequently encountered in Type A dissection that has extended to involve the descending aorta [similar to a Debakey Type I dissection]). This is important, because inadvertent administration of a thrombolytic agent to a patient who seems to be having a "stroke" or an "MI" would be disastrous.
4. Blood pressure findings
   a. Normal or decreased → ascending aorta
   b. Increased → descending aorta
   c. A difference in extremity blood pressure of 20 mmHg
5. Shock-like appearance (cool, clammy skin) despite increased blood pressure
6. Focal neurologic deficits
7. Unequal or absent pulses (hallmark of aortic dissection) between extremities
8. Diastolic murmur of aortic insufficiency (with Types I, II, and A)
9. Signs of cardiac tamponade (with Types I, II, and A)

F. Diagnostic evaluation
1. Initial studies
   a. Chest radiograph
      (1) Should be taken immediately
      (2) Patient should be in upright position.
      (3) Usually abnormal but findings are nonspecific; a normal chest radiograph does not exclude the diagnosis.
      (4) Suggestive findings
         (a) Mediastinal widening >8 cm (most common finding)
         (b) A change in the configuration or size of the thoracic aorta when compared with older radiographs; loss of space between the aorta and pulmonary artery
         (c) "Eggshell" or "calcium" sign → extension of the aortic shadow by >5 mm beyond its calcified aortic wall (when present, is pathognomonic for dissection)

(d) A blurred aortic knob or one with a localized hump

(e) A "double density" appearance of the aorta, which suggests the presence of true and false channels; the false lumen is less radiopaque than the true lumen.

(f) On the right side of the radiograph

i. Deviation of the trachea/nasogastric tube

ii. Shift and elevation of the right mainstem bronchus

iii. Deviation of the right paraspinous line

(g) On the left side of the film

i. A pleuro-apical cap

ii. Depressed left mainstem bronchus

iii. Pleural effusion

b. ECG

(1) Abnormal in most patients with aortic dissection

(2) Changes consistent with acute ischemia have been reported in up to 40% of cases (the most common misdiagnosis in these patients). STEMI ECG patterns may occur in up to 8% of cases and usually involve the inferior leads, because they involve the right coronary artery. A dissection-induced MI should be excluded (with transesophageal echocardiography or CT) in patients who are candidates for thrombolytic therapy and have clinical findings that suggest the possibility of thoracic aortic dissections.

(3) Varying degrees of AV block can be produced by propagation of the dissection into the ventricular septum.

(4) Left ventricular hypertrophy (due to long-standing hypertension) is a frequent finding.

2. Definitive studies: one (or more) of the following tests may be used to confirm the diagnosis. Each has certain advantages and disadvantages (and their 24-hour availability is variable). The diagnostic approach chosen will vary from institution to institution and patient to patient depending on the technology available, the speed of obtaining each diagnostic test, the experience of the radiologist in interpreting each test, the preference of the surgeon, the probability of the diagnosis, and comorbid disease of the patient. When the probability of dissection is high, the most readily available study should be the initial study of choice.

a. Transesophageal echocardiography (with color-coded Doppler imaging)

(1) Most expedient technique for confirming the diagnosis of aortic dissection; the diagnostic study of choice in many hospitals

(2) Sensitivity and specificity nearly 100%

(3) All the information required for decision-making regarding emergency surgical intervention can be obtained with this study; in addition to identifying the dissection, it detects the intimal tear site, flow in the proximal coronary arteries, aortic regurgitation, and pericardial effusion; it also differentiates true from false lumens.

(4) Advantages: only minimally invasive, requires only 10–15 minutes to complete, does not necessitate exposure to IV contrast, and can be performed at the bedside in the emergency department while the patient is being treated and appropriately monitored.

(5) Disadvantages: contraindicated in patients with esophageal disease (eg, strictures, varices), and the necessary equipment/expertise are not readily available in all hospitals.

b. Aortography
   (1) The traditional diagnostic gold standard
   (2) Sensitivity and specificity ~90%
   (3) In addition to confirming the diagnosis, it defines the intimal tear site, the extent of the dissection, and assesses the aortic valves and branches.
   (4) Disadvantages: invasive, time-consuming, requires special personnel, cannot be performed in the emergency department, necessitates exposure to IV contrast, is not 100% accurate (can miss dissection if the false lumen is thrombosed)
   (5) Best reserved for situations in which transesophageal echocardiography is not available or is nondiagnostic

c. MRI
   (1) Sensitivity and specificity 100%
   (2) Delineates the desired anatomy: type and extent of dissection, site of the intimal tear, presence of aortic insufficiency, and differential flow velocities in the true and false channels and in aortic branch vessels
   (3) Advantages: noninvasive, does not require IV contrast material or ionizing radiation
   (4) Disadvantages: time-consuming (requires >60 minutes to perform), cannot be performed in the emergency department or on patients with metallic implants, allows only limited access to the patient during the examination, some patients cannot tolerate the scanner, most units cannot accommodate monitoring or advanced life support equipment

d. Rapid-sequence (dynamic) CT scanning with contrast
   (1) Helical CT scans have almost 100% specificity and sensitivity; they are becoming the preferred diagnostic technique.
   (2) Can confirm the diagnosis, define the extent of the dissection, and distinguish between Type A and Type B dissections but cannot identify aortic insufficiency or extension to the aortic branches
   (3) Advantages: less invasive and provides greater contrast resolution than aortography, may reveal other abnormalities in cases in which dissection is excluded
   (4) Disadvantages: time-consuming, cannot be performed in the emergency department, exposes the patient to IV contrast, and may miss a dissection flap (if it is moving rapidly)
   (5) A good choice for emergency departments without access to transesophageal echocardiography because of its high sensitivity

G. Management

1. **All patients require 10–15 units of blood on stand-by and immediate consult with a thoracic surgeon.**
2. **Initial management for all types of dissections is medical and involves controlling the forces that propagate the dissection: pulse rate, systolic blood pressure, and rate of elevation of the aortic pulse pressure (dP/dT). If suspicion of dissection is high, begin treatment immediately; do not delay to await confirmatory study results.**
   a. **IV ß-blockers (eg, propranolol, metoprolol, atenolol, or esmolol) are administered first and titrated to a heart rate of 60–80 beats per minute.**
   b. **Nitroprusside is then started and titrated to a systolic blood pressure of 100–110 mmHg.**
3. **Hypotensive patients should be managed with small boluses of a crystalloid solution or blood.**

4. **Pain should be treated with IV narcotics.**
5. **Long-term management**
   a. **Dissections involving the ascending aorta (Types A, I, and II) are treated surgically.**
   b. **Dissections involving only the descending aorta (Types B and III) are treated medically unless complications are present.**

II. ABDOMINAL AORTIC ANEURYSMS (AAA)

A. Definition and anatomic location

1. Aortic aneurysms are true aneurysms (they involve all three layers of the arterial wall).
2. The vast majority (97%) are infrarenal in location.

B. Pathogenesis

1. Although AAAs often occur in patients with atherosclerotic disease, atherosclerosis is no longer believed to be the primary etiologic factor in the development of these aneurysms.
2. They are currently theorized to be due to a combination of genetic, structural, and metabolic factors including:
   a. Genetic predisposition
   b. Increased levels of elastase and collagenase
   c. Failure or loss of blood vessel elastin
   d. Copper deficiency
   e. Infection (mycotic aneurysms)
   f. Inflammatory disorders
   g. Local mechanical forces

C. Risk factors

1. **Advanced age (75% of AAAs are in patients >60 years old)**
2. **Male sex (particularly white males)**
3. **Family history of an AAA in a first-degree (blood) relative**
4. **Smoking history**
5. **Hypertensive history**
6. **History of coronary artery disease or peripheral vascular disease**
7. **Increased serum cholesterol levels**

D. Clinical presentation

1. Frequently a middle-aged or older man (and a heavy smoker) who had a syncopal episode at home with transient improvement
2. Sudden onset of severe abdominal, back, or flank pain with or without an associated syncopal episode (due to sudden hemorrhage) is the classic presentation: retroperitoneal bleeding with hematoma formation (usually on the left) occurs in most patients and accounts for the variety of clinical presentations that are possible and confusing.
   a. Flank pain (usually on the left side) may radiate to the groin and be accompanied by hematuria, thus simulating a kidney stone (the most common misdiagnosis).

b. Low back pain may be dull and radiate into the legs, thus mimicking musculoskeletal back pain.

c. Abdominal pain that is localized to the left lower quadrant and accompanied by guaiac-positive feces are common in patients with diverticulitis, but it is also seen in a ruptured AAA (and may be associated with a nonpulsatile mass).

d. A scrotal hematoma can be interpreted as a "mass" in the scrotum and thus simulate an incarcerated hernia.

e. Ecchymoses can result from significant bleeding and may be seen on the abdominal wall, flank, scrotum, penis, inguinal region, perineum, or perianal area.

f. Femoral neuropathy (pain in the hip and thigh, quadricep muscle weakness, diminished sensation over the anteromedial thigh and a weakened patellar reflex) may be from femoral nerve compression due to a hematoma.

3. Physical examination findings are variable.

a. A pulsatile (and occasionally tender) mass is palpable in the epigastric area in 77% of patients with a ruptured AAA; unruptured aneurysms are smaller in size and less frequently detected; in the presence of obesity or abdominal distention, a pulsatile mass may not be felt.

b. A tender (pulsatile) mass is highly suggestive of a rapidly expanding or recently ruptured AAA; most intact aneurysms are not tender.

c. Bruits may be heard over the abdominal aorta or femoral arteries.

d. Signs of distal extremity ischemia (not always present): unequal or unsynchronized distal pulses and cool, pale skin.

E. Diagnosis and management

1. **Presence of a symptomatic AAA is usually suggested by the clinical presentation and physical examination. This information alone is sufficient evidence in many patients to take them immediately to the operating room (particularly the hemodynamically unstable ones).**

2. **Once this diagnosis is suspected, the following measures should be undertaken immediately:**

a. **Two large-bore IV lines (normal saline or lactated Ringer's), cardiac monitor, pulse oximeter, supplemental oxygen**

b. **Draw blood for preoperative laboratory studies, and type and cross for at least 10 units of blood.**

c. **Order an ECG.**

d. **Obtain immediate surgical consult (preferably with a vascular surgeon).**

3. **Further evaluation is determined by the hemodynamic stability of the patient.**

a. **Hemodynamically unstable patients with a suspected AAA require prompt surgical repair. Fluid and blood resuscitation should be initiated in the emergency department, and the patient should be transferred to the operating room for definitive treatment as soon as the surgeon is available. Any delay for further diagnostic testing increases the risk of death from exsanguination.**

b. **Hemodynamically stable patients in whom this diagnosis is suspected may undergo further diagnostic testing (under close medical supervision) before surgical intervention. The various imaging modalities available are described below. Of these, ultrasonography or CT is the most appropriate studies in this setting.**

(1) Plain abdominal radiographs (AP and lateral films): although most patients with an AAA (75%) have suspicious findings on plain radiographic evaluation (aneurysmal calcification, soft-tissue mass, loss of renal shadow, renal displacement, and/or change in the posterior peritoneal flank stripe) except for aneurysmal calcification, none of these findings is specific for AAA and their absence does not reliably exclude the presence of an AAA. Furthermore, plain radiographs are not very accurate in determining the extent of the aneurysm, rupture, or associated vascular pathology.

(2) Ultrasonography has a sensitivity of nearly 100% in detecting an AAA and has the advantage of being quick, readily available, noninvasive, and portable. It cannot, however, accurately detect aneurysmal leaking and complications such as visceral or renal artery involvement. Moreover, the study may be limited in the presence of obesity or intestinal gas. Thus, ultrasound is most useful in determining whether or not an aneurysm is present, which may be more than enough information in the appropriate setting.

(3) CT with contrast also has a sensitivity of ~100% in detecting an AAA but is more accurate than ultrasound in detecting aneurysmal rupture and visceral artery involvement; it is not limited by obesity or intestinal gas. Visualization of the retroperitoneal structures and diagnosis of other pathology are also superior with this modality. Disadvantages include exposure to IV contrast material and longer study time.

(4) MRI: excellent at imaging the aorta and is superior to CT in assessing branch vessel involvement. However, it is of limited use in this setting because it is time consuming, has limited availability, cannot be performed in the emergency department or in the patient with metallic implants, and allows only limited access to the patient during the study. This modality is usually reserved for evaluation of asymptomatic patients.

(5) Angiography: although useful in determining the anatomy of the aorta and demonstrating occlusive lesions, angiography is not entirely reliable in detecting the presence (high false negative rate) or diameter of an AAA. Furthermore, it is time consuming, invasive, requires specialized personnel, and necessitates exposure to IV contrast. For these reasons, angiography is the least desirable study for emergent diagnostic evaluation of these patients.

DEEP VENOUS THROMBOSIS (DVT)

I. PATHOGENESIS

DVTs have their origin from Virchow's triad of venostasis, hypercoagulability, and vessel wall injury/abnormality.

II. CLINICAL PRESENTATION

A. Acute DVT

1. The physical manifestations of DVT are determined by the degree of thrombosis present (partial versus totally occluding), its location, and the extent of collaterals at the level of the occlusion. Physical findings may be minimal or absent and cannot be relied on by themselves to make or exclude the diagnosis.
2. Common signs and symptoms
   a. Unilateral pain, swelling, edema (most reliable sign), and tenderness
   b. Presence of a palpable cord (most often detected in the popliteal fossa), superficial venous dilatation, discoloration, and Homans sign (the least reliable finding)
3. Unilateral swelling is the most specific physical finding (especially if the measured difference is >3 cm).
4. Tenderness, erythema, or induration in the groin and popliteal fossa (where the femoral and popliteal veins are quite superficially located) are also highly suggestive of acute thrombosis of the underlying vessel.

B. Massive DVT: an ischemic form of venous occlusion due to massive iliofemoral thrombosis that also involves most of the venous collateral system

1. Phlegmasia cerulea dolens (painful blue inflammation)
   a. Occurs in <1% of patients with symptomatic venous thrombosis
   b. The leg is tensely swollen, painful, and cyanotic; petechiae and skin bullae may also be present.
   c. Occasionally results in venous gangrene (irreversible ischemia)
2. Phlegmasia alba dolens (painful milk/white leg)
   a. Due to massive iliofemoral thrombosis associated with arterial spasm
   b. The entire leg is swollen but not tense, and the pulse may be diminished.
   c. The skin is doughy and white; petechiae are often present.
   d. This is a temporary condition; as the arterial spasm resolves, the leg takes on the cyanotic appearance of phlegmasia cerulea dolens.

III. RISK FACTORS

A. The prevalence of DVT is directly correlated with the number of risk factors present; the greater the number of risk factors, the greater the risk of DVT.

B. Patients can be stratified into high- and low-risk groups based on the number of risk factors they possess, which can facilitate decision making
   1. Low-risk patients are those with one or no risk factors plus equivocal findings on physical examination.
   2. High-risk patients are those with multiple risk factors and highly suggestive examination findings.

C. Risk factors (Note: highest incidence of DVT is in patients undergoing surgical repair of a fractured hip.)
   1. Prior DVT: highest risk
   2. Carcinoma
   3. Age >40 years old
   4. MI/CHF/stroke
   5. Obesity
   6. Estrogen therapy
   7. Polycythemia vera
   8. Pregnancy or postpartum state
   9. Immobility or prolonged bed rest
   10. Recent trauma (including burns) or surgery
   11. Inherited abnormalities of coagulation (deficiency of antithrombin III, protein C, or protein S) or fibrinolysis
   12. Catheter placement (central venous, Swan-Ganz) and IV drug abuse
   13. Thrombocytosis
   14. Immune (eg, AIDS) and autoimmune (eg, systemic lupus erythematosus)

IV. PREDICTING PRETEST PROBABILITY FOR DVT: THE WELL'S CLINICAL CRITERIA

Table 5: Well's Clinical Prediction Rule for DVT

Active cancer (palliative, within 6 months or ongoing treatment)	1
Paralysis, paresis, or recent immobilization	1
Recently bedridden (>3 days) or major surgery (within 4 weeks)	1
Localized tenderness along deep venous system	1
Entire leg swollen	1
Calf swelling >3 cm when compared with opposite leg (10 cm below tibial tuberosity)	1
Pitting edema (greater in suspected leg)	1
Collateral superficial veins (nonvaricose)	1
Alternative diagnosis as likely as (or greater than) DVT	–2

Total score interpretation

≥3 = high pretest probability (>65% risk)
1–2 = moderate pretest probability (17 % risk)
≤0 = low pretest probability (<5% risk)

V. DIAGNOSTIC EVALUATION

A. Diagnostic testing options

1. **Duplex venous ultrasonography (8-mode, ie, two-dimensional)**
   a. **Initial diagnostic test of choice in most institutions where it is available; the ideal study for evaluating pregnant patients in their first trimester as well as in those with renal insufficiency, diabetes, or an allergy to contrast dye.**
   b. **Combines real-time ultrasonographic two-dimensional imaging with simultaneous Doppler flow evaluation with color-flow mapping, a process called "duplex scanning" (which is the best method of differentiating cellulitis from DVT).**
   c. **Advantages**
      (1) **Noninvasive and inexpensive**
      (2) **Sensitivity of 93% and specificity of 98% for detecting proximal DVTs**
      (3) **Identify other causes of calf pain and swelling (Baker cyst, abscess, or hematoma)**
   d. **Disadvantages**
      (1) **Less sensitive than venography in detecting isolated calf DVT and thrombosis above the groin, ie, pelvic or inferior vena caval thrombosis**
      (2) **Less accurate in the second and third trimesters of pregnancy**
   e. **Reliability**
      (1) **In patients with a low pretest probability, a single, negative, lower extremity venous ultrasound scan is sufficient to exclude "clinically significant" proximal DVT.**

(2) Patients with a moderate to high pretest probability require serial ultrasound scans if the initial one is negative.

2. Contrast venography
   a. Formally the accepted standard against which all other diagnostic tests are measured
      (1) Largely been replaced by duplex ultrasonography
      (2) Now used primarily in patients with uncertain ultrasound results
   b. Advantages
      (1) Sensitivity and specificity of nearly 100%
      (2) Outlines the entire venous system
   c. Disadvantages
      (1) Invasive, painful, expensive, limited in availability, and associated with risk of contrast-related allergic reactions and postvenography phlebitis/DVT
      (2) One-quarter of patients with suspected DVT have contraindications to venography (renal failure, dye allergy, pregnancy) or nondiagnostic studies.
3. MRI
   a. Detects calf, thigh, pelvic, renal, and pulmonary thrombi; can also diagnose other anatomic causes of leg pain and swelling
   b. Advantages
      (1) Noninvasive
      (2) No ionizing radiation or IV contrast
      (3) 97% sensitive and 95% specific for DVT
   c. Disadvantages
      (1) Expensive, not readily available, and requires significant patient cooperation
      (2) Should not replace ultrasound as the primary screening tool
   d. Most useful during the second and third trimesters of pregnancy when ultrasound is less accurate
4. D-dimer assay
   a. D-dimer
      (1) Fibrin degradation product, which is increased in patients with DVT and pulmonary embolism
      (2) Increased in many conditions (poor specificity) such as acute MI, stroke, trauma, and pregnancy
   b. There are a number of rapid D-dimer assays, including qualitative whole blood agglutination tests (SimpliRED) and quantitative turbidimetric or ELISA tests, in which levels >500 mg/L are abnormal.
   c. Like duplex ultrasonography and impedance plethysmography, it is more sensitive for proximal clots (93%) than for distal ones (70%).

B. Diagnosis

1. DVT should be suspected from the patient's signs, symptoms, and history of risk factors. However, a clinical diagnosis of DVT is insensitive and inaccurate; therefore, the diagnosis must be confirmed (with one of the diagnostic tests described above) before treatment.

2. The current recommended diagnostic algorithm is to use a combination of a clinical scoring system (eg, Well's Criteria, see Table 5) to determine pretest probability in conjunction with a D-dimer assay and/or venous ultrasound.
3. For patients at low risk of DVT
   a. A negative D-dimer excludes DVT.
   b. A positive D-dimer warrants venous ultrasound. If this is negative, DVT is excluded. If positive, proceed with anticoagulation.
4. For patients at moderate or high risk of DVT
   a. Initially order venous ultrasound. If positive, anticoagulate.
   b. However, a negative ultrasound in a predicted moderate- or high-risk patient does not exclude DVT. It is recommended to then order a D-dimer to further risk stratify these higher-risk patients.
      (1) If the D-dimer is negative, a DVT is excluded, and no further testing is required.
      (2) If the D-dimer is positive, the patient requires a repeat ultrasound in 2–7 days.
5. The treatment of isolated calf DVT remains controversial, even though ~25% of these thrombi propagate proximally and may subsequently embolize. If an isolated calf DVT is detected, the risk and benefit of anticoagulation should be weighed against patient's pretest probability.
   (1) For moderate- and high-risk patients, anticoagulation should be started unless contraindications exist.
   (2) For low-risk patients, repeat ultrasound is required with either no treatment or simply starting aspirin therapy.

VI. PHARMACOLOGIC THERAPY

A. Goals

1. Prevent pulmonary embolism
2. Reduce morbidity associated with the acute event
3. Prevent (or minimize) postphlebitic syndrome

B. Anticoagulation

1. IV heparin (unfractionated) or low-molecular-weight heparin (LMWH) are first-line options for a patient with DVT. However, LMWH and unfractionated heparin are contraindicated in patients with a history of heparin-induced thrombocytopenia; these patients should receive alternative agents (danaparoid, lepirudin, or argatroban). Four LMWHs are approved for therapy:
   a. Enoxaparin
      (1) 1 mg/kg SC every 12 hours (inpatient or outpatient)
      (2) 1.5 mg/kg/day SC (inpatient only)
   b. Tinzaparin 175 units/kg/day SC
   c. Dalteparin
      (1) 100 units/kg SC every 12 hours (inpatient or outpatient)
      (2) 200 units/kg/day SC (inpatient only)

d. Nadroparin
   (1) 86 units/kg SC every 12 hours (inpatient or outpatient)
   (2) 171 units/kg/day SC (inpatient only)

2. Fondaparinux
   a. An antithrombotic agent that is a specific inhibitor of Factor X
   b. As effective as enoxaparin in the treatment of DVT
      (1) Advantage: once-a-day dosing (enoxaparin is twice a day)
      (2) Disadvantage: no specific antidote for bleeding complications (although it is considered as safe as enoxaparin)
      (3) 7.5–10 mg/day SC
3. Patients with antithrombin III deficiency require pretreatment with antithrombin III concentrate or fresh frozen plasma to replenish this factor before heparinization.
4. Warfarin 5 mg/day should generally be started on day one of treatment; anticoagulation parameters should be checked on day three.
5. Outpatient treatment may be considered for carefully selected patients.
   a. No serious concomitant disease requiring hospitalization
   b. Communication and transportation is adequate
   c. INR blood level checked in 2–3 days

C. Thrombolytic therapy

1. Compared with heparin therapy, thrombolytic therapy resolves symptoms more rapidly, preserves valve integrity, and may decrease the incidence of postphlebitic syndrome. However, indications for thrombolytics in patients with DVT are unclear.
2. Usually reserved for patients <60 years old with massive or limbthreatening iliofemoral thrombosis (such as those with phlegmasia cerulea dolens) and for those with upper extremity DVT who have had symptoms <1 week and who have a low risk of bleeding.
3. Before thrombolytic therapy is administered, the diagnosis must be confirmed with an objective diagnostic study, and contraindications must be excluded (see discussion of reperfusion therapy in MI, pages 57–59).
4. Administered in conjunction with heparin therapy
5. Associated with an increased risk of bleeding (about three times greater than heparin, which is greater than the risk of bleeding associated with the use of thrombolytic agents for treatment of acute MI)
6. Possible agents include streptokinase, urokinase, and tissue plasminogen activator.

D. Inferior vena caval interruption with a Greenfield filter

1. Primary indication
   a. Proximal DVT (above the level of the knee) in a patient with a contraindication to anticoagulation or thrombolytic therapy
   b. Those who require urgent surgery that precludes anticoagulation or those in whom treatment has not been effective
2. Other indications
   a. Recurrent DVT despite adequate anticoagulation
   b. Presence of a large free-floating caval thrombus
   c. Chronic recurring embolization in a patient with pulmonary hypertension
3. Patients with malignancies seem to benefit most from this procedure.

VII. DISPOSITION—ADMISSION CRITERIA

A. Extensive ileofemoral DVT with circulatory compromise

B. Increased risk of bleeding requiring close monitoring of therapy

C. Limited cardiorespiratory reserve

D. Risk of poor compliance with home therapy or inadequate assistive support

E. Contraindications to LMWH heparin necessitating IV heparin therapy

HYPERTENSIVE EMERGENCIES AND URGENCIES

I. HYPERTENSIVE EMERGENCIES

A. Definition

1. Patients with severely increased diastolic blood pressure (>140 mmHg) and evidence of acute end-organ dysfunction or damage
2. The presence of end-organ damage (not the absolute blood pressure) determines a patient with a hypertensive emergency.
3. The heart, brain, and kidneys are the organs most often affected.

B. Clinical presentation

1. Malignant hypertension
2. Hypertensive encephalopathy
3. Hypertension with acute intracranial events (hemorrhagic stroke, thrombotic stroke, subarachnoid hemorrhage)
4. Aortic dissection
5. Acute pulmonary edema
6. Acute myocardial ischemia or injury
7. Eclampsia
8 Acute hypertensive renal insufficiency
9. Catecholamine-induced hypertensive crisis: an acute increase in circulating catecholamines that produces an increased blood pressure with headache, palpitations, sweating, and tachycardia. Causes include:
   a. Pheochromocytoma (adrenal tumor)
   b. Concomitant use of monoamine oxidase inhibitors and sympathomimetic agents. Commonly implicated sympathomimetics include:
      (1) Tyramine (Chianti wine, aged cheese, beer, pickled herring, chicken liver)
      (2) Ephedrine and phenylpropanolamine (found in OTC cold and cough preparations and appetite suppressants)
   c. Acute cocaine intoxication
   d. Acute clonidine withdrawal (particularly when withdrawn simultaneously with a ß-blocking agent)

C. Management

1. The goal of therapy is to arrest and reverse the progression of endorgan dysfunction while maintaining organ perfusion and avoiding complications. To do this, the blood pressure must be lowered rapidly and in a controlled manner. The exact extent of blood pressure reduction is determined by the clinical situation; however, a reasonable target is to reduce the blood pressure by 20%–30% of pretreatment levels over the first hour of therapy.
2. Medications are usually given by the IV route, which provides a faster onset of action and is more easily titrated.
3. The antihypertensive agent of choice varies with the specific hypertensive emergency.

a. Malignant hypertension and hypertensive encephalopathy: Historically, nitroprusside was the drug of choice. However, IV labetalol is now used most commonly, particularly when intra-arterial pressure monitoring is not available. Nicardipine IV is also effective.

b. Hypertension with an ischemic stroke: treatment of hypertension in the immediate poststroke period can be detrimental in these patients and should not be initiated in most instances; acute reduction in blood pressure can reduce perfusion to the surrounding watershed areas and may thereby extend the infarction. Therefore, blood pressure reduction should be considered only in patients with extremely high blood pressures (>220/120 mmHg). Short-acting agents with few CNS effects (such as IV nitroprusside or IV labetalol) are the drugs of choice.

c. Hypertension with hemorrhagic stroke: although somewhat more controversial, most authors agree that mild to moderate hypertension is generally well-tolerated in these patients, but that severe hypertension (blood pressure > 220/120 mmHg) may promote further hemorrhage and should be judiciously controlled with a titratable agent such as IV labetalol (other acceptable agents: esmolol, enalapril, nicardipine).

d. Hypertension with subarachnoid hemorrhage: nimodipine or nicardipine (calcium channel blockers) is the initial therapeutic agent of choice. In addition to its antihypertensive effect, nimodipine also decreases the cerebral vasospasm that occurs after subarachnoid hemorrhage. The goal of treatment is to reduce the mean arterial pressure to prehemorrhagic levels. Blood pressure reduction is associated with a decrease in the risk of rebleeding in these patients.

e. Aortic dissection: nitroprusside in combination with an IV ß-blocker (propranolol or esmolol) is the therapy of choice; the ß-blocking agent should be administered first to prevent the reflex tachycardia that can occur in association with nitroprusside. IV labetalol as a single drug therapy is the alternative agent of choice.

f. Acute myocardial ischemia or injury: IV nitroglycerin is the primary agent of choice because of its beneficial effects on coronary perfusion. However, an IV ß-blocker should also be given to reduce myocardial oxygen demand. If blood pressure remains increased despite aggressive use of nitroglycerin and ß-blocker, nitroprusside is the alternative of choice; however, it can increase myocardial ischemia via the coronary steal syndrome.

g. Acute pulmonary edema: if standard measures for pulmonary edema (eg, noninvasive positive pressure ventilation, high-dose IV nitroglycerin, IV furosemide) fail to adequately decrease blood pressure, specific antihypertensive therapy should be initiated. IV nitroglycerin is the drug of choice. If blood pressure remains increased despite aggressive use of nitroglycerin, nitroprusside is the alternative agent.

h. Eclampsia: IV magnesium sulfate in combination with IV hydralazine (available as a generic) are first-line agents. IV labetalol is also safe and effective. Nitroprusside may be used in postpartum eclampsia but, because of the risk of fetal cyanide poisoning, is relatively contraindicated antepartum; it should be reserved for those patients who do not respond to the above-mentioned treatment regimens.

i. Acute hypertensive renal insufficiency: nitroprusside is the drug of choice, but patients must be carefully monitored for thiocyanate toxicity (which is more common in this setting). Nifedipine or labetalol are good alternatives.

j. Catecholamine-induced hypertensive crisis due to:

   (1) Pheochromocytoma or monoamine oxidase inhibitor interaction: IV labetalol as a single agent or phentolamine followed by an IV ß-blocker are first-line agents. Nitroprusside followed by an IV ß-blocker is also effective.

(2) Acute clonidine withdrawal: restart clonidine; IV labetalol or phentolamine followed by an IV ß-blocker is also effective.

(3) Acute cocaine intoxication: sedation with a benzodiazepine such as diazepam is usually very effective. If hypertension persists despite adequate sedation, administer IV labetalol or phentolamine (followed by an IV ß-blocker if severe tachycardia is present).

4. Medications to avoid in hypertensive emergencies
   a. Malignant hypertension/hypertensive encephalopathy
      (1) ACE inhibitors
      (2) Clonidine
      (3) Pure ß-blockers
   b. Acute myocardial ischemia or injury/aortic dissection
      (1) Diazoxide
      (2) Hydralazine
      (3) Minoxidil
   c. Acute pulmonary edema
      (1) Diazoxide
      (2) Hydralazine
      (3) Minoxidil
   d. Eclampsia
      (1) Diuretics (furosemide, bumetanide, hydrochlorothiazide)
      (2) ACE inhibitors
      (3) Nitroprusside: relatively contraindicated antepartum because of possible cyanide toxicity
   e. Acute hypertensive renal insufficiency
      (1) Diazoxide
      (2) Pure ß-blockers as single agents
   f. Catecholamine-induced hypertensive crisis
      (1) Minoxidil
      (2) Pure ß-blockers as single agents

II. HYPERTENSIVE URGENCIES

A. Definition and etiology

1. A diastolic blood pressure ≥115 mmHg without evidence of end-organ dysfunction or damage
2. These patients are asymptomatic and usually present to the emergency department for an unrelated problem.
3. Most common in patients with chronic hypertension who are noncompliant with their medications

B. Management

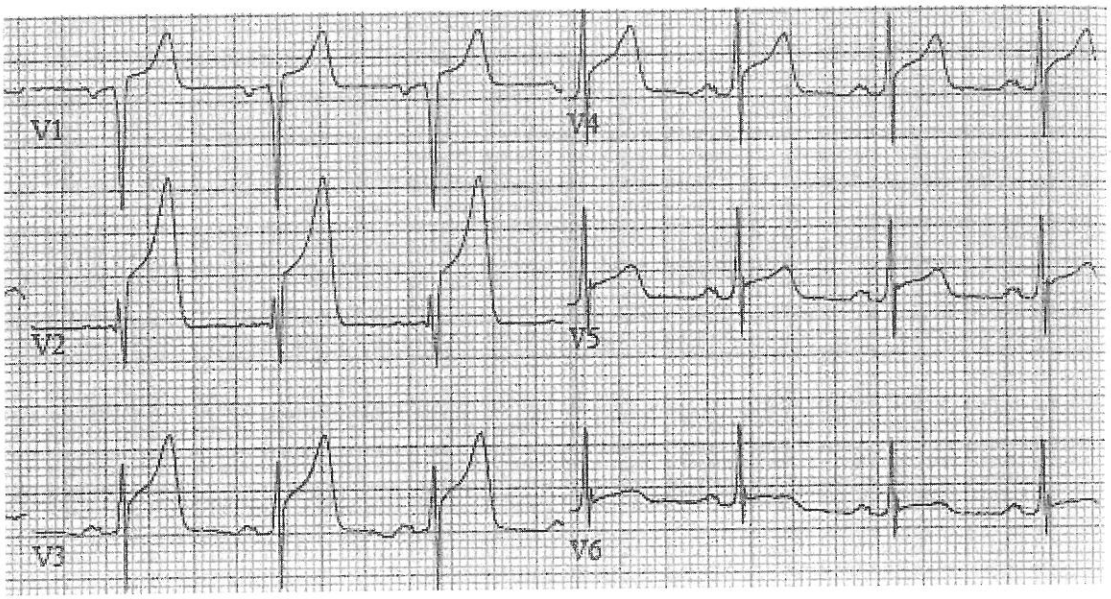
1. Goal of therapy is to lower the blood pressure gradually over a period of 24–48 hours with oral medications. *Catastrophic consequences may occur with IV administration.*
2. Commonly used agents
   a. Nifedipine
   b. Labetalol
   c. Clonidine
   d. ACE inhibitors

CARDIOVASCULAR DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A middle-aged man who has diabetes and hypertension presents with substernal, left-sided chest pain and a crushing/heavy pressure sensation, radiating to the left jaw and down the left arm. He appears diaphoretic and in obvious distress.



Courtesy of Dr. Michael McCrea

What is the diagnosis?

Scenario B

Presentation: A patient with acute decompensated heart failure presents with respiratory distress and gives a history that includes increasing lower extremity edema, orthopnea, paroxysmal nocturnal dyspnea, and a nonproductive cough.

Physical examination: The patient is in respiratory distress with hypoxia. Physical examination findings include jugular venous distention, rales on lung auscultation, and symmetrical, pitting lower extremity edema.

What is the diagnosis?

Scenario C

Presentation: An elderly patient with a history of uncontrolled hypertension presents with sudden onset of severe ripping, tearing chest pain radiating to the mid back between the scapulae. Since onset, the pain in the back has migrated caudally.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: STEMI

Diagnostic evaluation: The ECG shows ST-segment elevation in contiguous leads in an anatomic distribution (anterior).

Management: After IV access has been established, the patient should be placed on a monitor, provided low-flow supplemental oxygen for any dyspnea or hypoxia, and given aspirin 325 mg (chewed) unless he has a true aspirin allergy. Nitroglycerin can be administered as needed for chest pain. Coronary reperfusion is the definitive treatment and will be guided by availability of PCI. If the patient presents to a hospital with PCI capabilities, the cardiac catheterization team should be activated immediately and the patient prepared for emergent PCI with a first medical contact-to-device time of 90 minutes. If the patient had presented to a hospital that does not have PCI capabilities, the goal for "door-in, door-out" at the initial hospital is 30 minutes, with a first medical contact-to-device time of 120 minutes, allowing for transport time to the PCI hospital. The patient should also be given a heparin bolus followed by infusion.

Scenario B

Diagnosis: decompensated congestive heart failure

Diagnostic evaluation: Possible triggers for acute decompensation include medication noncompliance, dietary indiscretion (excess sodium intake), dysrhythmias, or acute coronary syndrome. Chest radiograph may show cardiomegaly depending on the cause of the heart failure. Chest radiograph will also show pulmonary venous congestion. An ECG should be obtained to evaluate for concurrent dysrhythmia or acute coronary syndrome. Laboratory studies should include renal function testing, cardiac biomarkers, and a brain natriuretic peptide.

Management: The patient should be started on BiPAP with supplemental oxygen if needed for hypoxia. ACE inhibitors and high-dose nitroglycerin should be given to lower afterload and preload, respectively. After this, if the patient is volume overloaded and has normal renal function, furosemide should be given for diuresis.

Scenario C

Diagnosis: thoracic aortic dissection

Diagnostic evaluation: Depending on the location of the dissection, there may be differences in the upper extremity blood pressures or a pulse difference between the right arm and left arm (Debakey I/II or Stanford A). A dissection below the origin of the left subclavian artery (Debakey III or Stanford B) would not have a difference in upper extremity pulses or blood pressures but would have a difference when comparing upper and lower extremities. An ECG should be done to exclude acute coronary syndrome as a cause of the chest pain or to evaluate for concomitant STEMI, because a

proximal dissection could involve the aortic root and coronary arteries. An initial chest radiograph should be obtained, because there are classic findings of aortic dissection on chest radiograph, most notably a widened mediastinum. However, the lack of classic radiographic findings does not exclude aortic dissection, especially in a patient with a classic presentation and risk factors as above. In stable patients, CTA of the chest or an echocardiogram (preferably transesphogeal) could be ordered depending on availability. Unstable patients should not have any additional imaging in the emergency department and should proceed to the operating room.

Management: Rapid control of heart rate and blood pressure is the primary treatment concern while arranging for definitive surgical care, especially in unstable patients. A ß-blocker should be given to decrease the heart rate, and a vasodilator such as nitroprusside should then be given for rapid control of blood pressure. The patient should be typed and crossmatched for 10 units of blood while preparing for the operating room.

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HEAD, EAR, EYE, NOSE, AND THROAT DISORDERS: SELF-ASSESSMENT QUESTIONS

1. All of the following are true of peripheral vertigo except:

   (a) Onset is gradual.
   (b) Nystagmus may be horizontal or horizontorotary in direction.
   (c) It may be associated with nausea, vomiting, hearing loss, and tinnitus.
   (d) It is generally self-limited.

2. The most reliable sign of acute otitis media is:

   (a) A tympanic membrane that is erythematous
   (b) The presence of fever
   (c) Loss of mobility of the tympanic membrane on pneumatic otoscopy
   (d) The presence of a scarred and retracted tympanic membrane

3. Which of the following statements regarding malignant otitis externa is true?

   (a) It is a common complication of otitis externa that afflicts otherwise healthy patients.
   (b) It is treated on an outpatient basis with oral antibiotics.
   (c) It is caused by *Pseudomonas*.
   (d) Patients with this disease process are generally afebrile and experience little pain.

4. Which of the following is least characteristic of croup?

   (a) Barking cough
   (b) High fever
   (c) Preceding upper respiratory infection
   (d) Insidious onset

5. All of the following are true of temporal arteritis except:

   (a) It is associated with a markedly increased sedimentation rate.
   (b) It may present as sudden, painless, unilateral loss of vision.
   (c) It is treated with steroids.
   (d) It is a nonsegmental vasculitis.

6. Appropriate antibiotic therapy of pharyngitis caused by group A ß-hemolytic streptococci can limit or prevent all of the following complications except:

   (a) Rheumatic fever
   (b) Glomerulonephritis
   (c) Pharyngeal space infections
   (d) Spread of the infection to others

7. _____ is/are the most common cause(s) of pharyngitis.

   (a) Group A ß-hemolytic streptococcus
   (b) Mycoplasma
   (c) Viruses
   (d) Gonorrhea

8. Which statement is most true regarding group A ß-hemolytic streptococcus pharyngitis?

   (a) Accurate diagnosis can be made on the basis of clinical findings alone.
   (b) It commonly affects children <3 years old.
   (c) Associated complaints of abdominal pain, vomiting, and headache are common in children.
   (d) Patients classically have tender posterior cervical adenopathy, mild fever, and exudative tonsillitis.

9. Which of the nerve blocks listed below is most appropriate when midfacial anesthesia is required?

   (a) Inferior alveolar nerve block
   (b) Infraorbital nerve block
   (c) Supraorbital nerve block
   (d) Posterior superior alveolar nerve block

10. All of the following statements regarding Ellis II fractures are true except:

    (a) They involve both the enamel and dentin.
    (b) They are associated with hot and cold sensitivity.
    (c) Bleeding from the tooth is characteristic.
    (d) Dental follow-up within 24 hours is recommended.

11. Which of the following descriptive statements is most consistent with alveolar osteitis?

    (a) It occurs within 24 hours of extraction and responds to oral analgesics.
    (b) It is seen several days after extraction, after an initial pain-free time interval, and is not relieved by oral analgesics.
    (c) It is associated with dental caries and treated by tooth extraction.
    (d) It is a periodontal lesion in which bacteria invade non-necrotic tissue.

12. Which is true regarding treatment of avulsed teeth?

    (a) The tooth should be wiped clean before replanting.
    (b) Primary teeth should be replaced immediately.
    (c) Handling the tooth by the root is preferred.
    (d) Hank's solution and milk are better transport solutions than tap water.

13. The most common arterial source of posterior epistaxis is:

(a) Sphenopalatine artery
(b) Anterior ethmoidal artery
(c) Posterior ethmoidal artery
(d) Kiesselbach plexus

14. All the following statements regarding posterior epistaxis are accurate except:

(a) It is less common than anterior epistaxis.
(b) It most commonly occurs in children and young adults.
(c) Placement of a posterior pack can result in hypoxia and hypercarbia.
(d) Patients treated with a posterior pack should be admitted.

15. The treatment of choice for simple group A ß-hemolytic streptococcus is:

(a) Penicillin
(b) Erythromycin
(c) Cephalosporins
(d) TMP-SMX

16. The following statements regarding glomerulonephritis are accurate except:

(a) It is a suppurative complication of group A ß-hemolytic streptococcus infection.
(b) It can result from either pharyngeal or cutaneous infection with group A ß-hemolytic streptococci.
(c) Patients usually present after a latent period of 1.5–3 weeks.
(d) The most useful laboratory test for making the diagnosis is the urinalysis.

17. The following statements regarding rheumatic fever are accurate except:

(a) The Jones Criteria (revised) are used to establish the diagnosis.
(b) Evidence of a preceding group A ß-hemolytic streptococcus infection plus the presence of one major and one minor criteria make the diagnosis highly probable.
(c) Patients usually present with severe migratory joint pain.
(d) Morbidity is most closely related to the development of carditis and valvular damage.

18. Bacterial tracheitis is most commonly caused by _____.

(a) *Haemophilus influenzae*
(b) *Streptococcus pneumoniae*
(c) *Staphylococcus aureus*
(d) Group A ß-hemolytic streptococci

19. A mother brings in her 3-year-old child for evaluation. She states that he was playing on the floor while she was preparing dinner and he suddenly started coughing. As you evaluate the child, you note that he is playful and in no acute distress. Which of the following statements is most accurate?

    (a) You should reassure the mother and send the child home; further evaluation is unnecessary, because foreign body aspiration is unlikely in this scenario.
    (b) You should obtain further history and perform PA and lateral chest radiographs; normal findings on these radiographs exclude the possibility of foreign body aspiration.
    (c) You should obtain further history and perform PA, lateral, and (possibly) bilateral decubitu chest radiographs; if these radiographs are negative (and your suspicion for foreign body aspiration remains high), bronchoscopy should be arranged.
    (d) You should immediately arrange for diagnostic bronchoscopy in every child with this presentation.

20. Which of the following is not true regarding peritonsillar abscesses?

    (a) It most commonly occurs as a complication of suppurative tonsillitis.
    (b) Needle aspiration provides sufficient initial drainage for most abscesses.
    (c) It is most common in infants.
    (d) The causative organism(s) is usually polymicrobial.

21. A child presents with complaint of decreased hearing after being slapped on the ear by his brother. Examination reveals a small perforation of the tympanic membrane and a conductiv hearing loss but is otherwise entirely normal. The child is acting normally, appears to be comfortable, and has normal vital signs. The most appropriate treatment is:

    (a) Emergent ENT referral for possible surgical repair.
    (b) A prescription for an oral antibiotic and referral for follow-up with ENT in 24 hours
    (c) A prescription for a topical antibiotic and referral for follow-up with ENT in 1 week.
    (d) Recommend the ear be kept dry, and refer to ENT on a nonurgent basis.

22. Which of the following statements regarding the diagnosis of sinusitis is most accurate?

    (a) It requires diagnostic imaging in most instances.
    (b) Normal findings on plain radiographs (Water, Caldwell, submental-vertex, and lateral views) excludes the diagnosis.
    (c) Plain films are the most useful in diagnosing sphenoid and ethmoid sinusitis.
    (d) The "gold standard" for diagnosing sinus disease and its complications is CT of the si

23. Which of the following statements regarding hairy leukoplakia is false?

    (a) It is a white plaque-like lesion with hairy projections.
    (b) It is usually asymptomatic.
    (c) It is easily removed with a tongue blade.
    (d) It is one of the most common virally induced, oral diseases of HIV-infected individu with a prevalance as high as 25%–50%.

24. The initial study of choice for confirming the presence of a retropharyngeal abscess is a soft-tissue lateral radiograph of the neck. To avoid obtaining a false-positive result, this film must be taken:

    (a) During expiration with the neck in slight extension
    (b) During expiration with the neck in slight flexion
    (c) During inspiration with the neck in slight extension
    (d) During inspiration with the neck in slight flexion

25. Croup (laryngotracheobronchitis) is most commonly caused by:

    (a) Respiratory syncytial virus
    (b) Parainfluenza virus
    (c) Adenovirus
    (d) Influenza virus

26. A 3-year-old is brought in for evaluation of sore throat, fever, and refusal to eat. The child's voice is muffled. Examination reveals a unilateral bulging of the posterior pharyngeal wall, tender anterior cervical adenopathy, and temperature of 102°F (39°C). The most likely diagnosis is:

    (a) Retropharyngeal abscess
    (b) Peritonsillar abscess
    (c) Ludwig angina
    (d) Masticator space abscess

27. An infant who was delivered at home and received no perinatal care is brought in 12 days after birth for evaluation of a purulent eye discharge and cough. Examination reveals diffuse conjunctival injection and normal pupillary reactivity. The most likely etiologic agent is:

    (a) *Staphylococcus aureus*
    (b) Adenovirus
    (c) *Chlamydia trachomatis*
    (d) *Neisseria gonorrhoeae*

28. A sexually active 20-year-old man presents with redness and irritation of his right eye with an associated discharge. Findings include diffuse conjunctival injection and a copious purulent ocular discharge. Visual acuity and pupillary reactivity are normal. The most likely etiologic agent is:

    (a) *Neisseria gonorrhoeae*
    (b) Herpes simplex
    (c) Adenovirus
    (d) Vernal conjunctivitis

29. The most appropriate therapy for a patient with conjunctivitis and corneal ulceration due to *Neisseria gonorrhoeae* is:

    (a) Discharge to home with topical erythromycin or tetracycline ophthalmic ointment
    (b) Discharge to home with tobramycin ophthalmic ointment or drops
    (c) Hospital admission with administration of IM or IV ceftriaxone
    (d) Hospital admission with administration of IV ceftriaxone, topical erythromycin, oral erythromycin (in children) or doxycycline (in adults), and saline irrigation

30. Which of the following entities is most characteristically associated with painless decrease (or loss) of vision?

    (a) Central retinal artery occlusion
    (b) Acute iritis
    (c) Acute narrow angle closure glaucoma
    (d) Optic neuritis

31. Which of the following is associated with pain and loss of central vision only?

    (a) Primary open angle closure glaucoma
    (b) Acute iritis
    (c) Acute anterior uveitis
    (d) Optic neuritis

32. A patient presents with eye pain, slight blurring of vision and severe photophobia. Examination reveals a red eye with a ciliary flush, a constricted pupil, and a clear cornea. Slit lamp examination reveals flare and cells in the anterior chamber. The most likely diagnosis is:

    (a) Acute angle closure glaucoma
    (b) Retrobulbar neuritis
    (c) Acute iritis
    (d) Primary open angle closure glaucoma

33. Which of the following is most appropriate in the treatment of acute traumatic iritis?

    (a) Oral steroid therapy
    (b) Topical steroids (in consultation with an ophthalmologist)
    (c) Antibiotic ointment or drops
    (d) Epsilon aminocaproic acid

34. The mechanism of action by which timolol and acetazolamide work in treatment of acute narrow angle glaucoma is:

    (a) They pull the iris back from its anterior position, thereby opening the angle and allowing escape of aqueous humor.
    (b) They decrease the secretion of aqueous humor by the ciliary body.
    (c) They cause pupillary block by dilating the iris and allowing the egress of aqueous humor.
    (d) None of the above

35. A 77-year-old woman presents with a headache and loss of vision in her left eye. Visual acuity is measured as bare light perception in this eye. The cornea is clear, pupillary reactivity is normal, and the sedimentation rate is 60. The most appropriate therapy for this patient is:

(a) Topical pilocarpine solution
(b) A topical steroid agent
(c) Ophthalmology referral for follow-up in 3-4 days
(d) High-dose corticosteroids, preferably IV

36. Which of the following is a common sign of a blowout fracture of the orbit with entrapment?

(a) Ptosis
(b) Exophthalmos
(c) Afferent pupil defect
(d) Pain and diplopia on upward gaze

37. A patient presents with a laceration to his upper eyelid. On evaluation, you note a deep horizontal laceration toward the middle of the upper lid and associated ptosis. The globe itself does not appear to be injured. The most likely associated injury is damage to the:

(a) Lacrimal canaliculi
(b) Orbital septum
(c) Levator muscle/tendon
(d) Canthal tendon

38. The finding most consistent with a ruptured globe:

(a) Teardrop or irregularly shaped pupil
(b) Increased intraocular pressure
(c) Exophthalmos
(d) Diminished intraocular pressure

39. The shortest-acting cycloplegic agent is:

(a) Cyclopentolate
(b) Tropicamide
(c) Homatropine
(d) Atropine

40. The topical antibiotic agent associated with the development of skin sensitivity in 10%–15% of patients is:

(a) Sulfacetamide
(b) Chloramphenicol
(c) Erythromycin
(d) Neomycin

41. Topical ophthalmic drops have color-coded caps to facilitate the identification of the various types of agents. The color cap associated with miotic agents is:

    (a) Green
    (b) Red
    (c) Yellow
    (d) White

42. The presence of a Marcus-Gunn pupil on examination is most indicative of:

    (a) Acute iritis
    (b) Optic neuritis
    (c) Vitreous hemorrhage
    (d) Functional blindness

43. A 70-year-old woman presents with obvious signs and symptoms of acute angle closure glaucoma. Her past medical history is significant for poorly controlled CHF. Which of the following would be inappropriate?

    (a) Pilocarpine 2% solution
    (b) Glycerol 50% solution
    (c) Timolol 0.5% solution
    (d) Acetazolamide

44. Which of the following statements regarding periorbital cellulitis is most accurate?

    (a) Children >10 years old are rarely affected.
    (b) Patients present with erythema, warmth, and swelling of one or both eyelids.
    (c) Patients complain of pain with ocular movement, and ophthalmoplegia may be present.
    (d) Fever is uncommon.

45. Which of the following statements regarding use of a tonometer is most accurate?

    (a) Its use is contraindicated in the presence of a ruptured globe.
    (b) Intraocular pressure is always high in acute iritis.
    (c) Use of an air-puff tonometer requires direct eye contact.
    (d) The Schiotz tonometer gives a direct reading in millimeters of mercury.

46. Which of the following conditions does not classically produce a decrease in visual acuity?

    (a) Traumatic lens dislocation
    (b) Retrobulbar hematoma
    (c) Periorbital cellulitis
    (d) Retrobulbar neuritis

47. A 25-year-old patient presents with a foreign body sensation in his left eye, photophobia, and tearing. Evaluation reveals a visual acuity of 20/30, diffusely reddened eye, decreased corneal sensation, and a dendritic lesion on fluorescein staining. Which of the following could produce rapid worsening of his condition and should not be prescribed?

(a) A topical antibiotic
(b) A topical steroid
(c) A topical antiviral agent (in consultation with an ophthalmologist)
(d) A mydriatic agent

48. A patient who uses extended-wear soft contact lenses presents with complaints of photophobia, eye pain, blurred vision, and a purulent discharge. Examination reveals a corneal ulcer. The causative agent in this setting is most likely:

(a) *Neisseria gonorrhoeae*
(b) *Klebsiella*
(c) Adenovirus
(d) *Pseudomonas*

49. Immediate ophthalmology consult, hospital admission and treatment is mandatory for which of the following conditions?

(a) Orbital (postseptal) cellulitis
(b) Herpes zoster ophthalmitis
(c) Corneal ulcers
(d) Acute iritis

50. Regarding chemical burns of the eye, which of the following statements is most accurate?

(a) Alkali burns are usually worse than acid burns.
(b) Alkali burns produce a coagulation necrosis.
(c) A neutralizing agent should be used to irrigate the eye.
(d) Irrigation can be discontinued after 30 minutes, once the pH of the conjunctival fornix is neutral at 8.5.

51. The most appropriate management sequence for an acid or alkali burn of the eye is:

(a) Detailed eye examination, assessment of visual acuity, copious irrigation, pH assessment
(b) Evaluation of visual acuity, pH assessment, copious irrigation, detailed eye examination
(c) pH assessment, copious irrigation, evaluation of visual acuity, detailed eye examination
(d) Copious irrigation, pH assessment, evaluation of visual acuity, detailed eye examination

52. A 42-year-old man presents with painful swelling below the inner aspect of his right eye of 1 day duration. Examination reveals localized erythematous swelling and tearing. His visual acuity is 20/20, the remainder of his eye examination is unremarkable, and he otherwise appears well. The most appropriate treatment is:

    (a) Immediate incision and drainage
    (b) Admission for parenteral antibiotics
    (c) A topical antibiotic ointment
    (d) A broad-spectrum oral antibiotic and warm compresses

53. A patient with AIDS and a CD4 count <100 presents with decreased vision in his left eye. Funduscopic examination reveals fluffy white perivascular retinal lesions associated with hemorrhage. The most appropriate treatment of this patient involves immediate ophthalmology consult and _____.

    (a) IV acyclovir
    (b) IV steroids
    (c) IV ganciclovir or foscarnet
    (d) Topical steroids

54. The most common cause of conjunctivitis is:

    (a) *Staphylococcus aureus*
    (b) *Chlamydia trachomatis*
    (c) *Neisseria gonorrhoeae*
    (d) A virus

55. Which of the following statements about viral conjunctivitis is most accurate?

    (a) Adenovirus is the most common offending agent.
    (b) Constitutional symptoms consistent with a viral syndrome are present in <10% of patients.
    (c) A follicular response of the palpebral conjunctiva and preauricular adenopathy are rare examination findings.
    (d) The associated discharge is typically mucopurulent.

56. Initial management for patients with primary open angle glaucoma usually consists of:

    (a) Topical medications to decrease intraocular pressure
    (b) Oral steroids to decrease intraocular pressure
    (c) Laser or surgical therapy
    (d) Observation and close follow-up only, until symptoms develop

ANSWERS

1. a	11. b	21. d	31. d	41. a	51. d
2. c	12. d	22. d	32. c	42. b	52. d
3. c	13. a	23. c	33. b	43. c	53. c
4. b	14. b	24. c	34. b	44. b	54. d
5. d	15. a	25. b	35. d	45. a	55. a
6. b	16. a	26. a	36. d	46. c	56. a
7. c	17. b	27. c	37. c	47. b	
8. c	18. c	28. a	38. a	48. d	
9. b	19. c	29. d	39. b	49. a	
10. c	20. c	30. a	40. d	50. a	

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

OTOLOGIC EMERGENCIES

I. ANATOMY

A. Sensory supply to the ear

1. Tympanic membrane: auriculotemporal (V3), cranial nerves IX and X
2. Auricle: auriculotemporal (V3), cranial nerves XII and X, lesser occipital, greater auricular
3. External auditory canal: auricolutemporal (V3), cranial nerves VII, IX, and X

B. Auditory transmission

Sound waves enter ear canal → tympanic membrane → ossicles → perilymph → endolymph → organ of Corti → spiral ganglion → cochlear nulei → temporal lobes

II. INFECTIONS

A. Acute otitis externa (swimmer's ear)

1. ***Pseudomonas aeruginosa*** **and** ***Staphylococcus aureus*** **are responsible for most cases of otitis externa. Other pathogens include** ***Proteus***, ***Streptococcus***, **and fungi (usually** ***Aspergillus***).
2. **A major predisposing factor is exposure to a warm, moist environment, so most cases occur in the summer months and in patients exposed to tropical environments; foreign body trauma and water exposure are obvious risk factors.**
3. **Clinical presentation**
   a. **Itching: may be intense with severe fungal infections**
   b. **Pain: may be intense, constant, and aggravated by pulling on the ear or pressing on the tragus**
   c. **Sense of fullness in the ear**
   d. **Purulent discharge**
   e. **External auditory canal is often edematous and erythematous with significant debris.**
   f. **If the tympanic membrane can be visualized, it is sometimes red, thick, and covered with flat vesicles or areas of desquamating epithelium.**
4. **Differential diagnosis**
   a. **Bacterial acute otitis media**
   b. **Fungal acute otitis media (otomycosis)**
   c. **Suppurative otitis media with tympanic membrane perforation**
   d. **Contact dermatitis (eg, neomycin)**
   e. **Eczema**
   f. **Psoriasis**
5. **Management**
   a. **Cleanse the ear canal of debris using suction, irrigation, or gentle curettage; this is the most important part of therapy.**
   b. **Use topical agent**

D. Acute otitis media

1. Epidemiology
   a. Children 6 months to 6 years old are most often affected; incidence peaks at 6–18 months of age.
   b. More common in the winter and spring months; frequently occurs in association with a viral upper respiratory infection
2. Pathophysiology
   a. Eustachian tube dysfunction plays a central role in pathogenesis.
   b. Eustachian tube dysfunction → retention of secretions → colonization (usually bacterial, although antibiotics are not always necessary)
3. Etiology — the most common bacterial pathogens:
   a. *Streptococcus pneumonia* (most common in all age groups), *Haemophilus influenzae, Moraxella catarrhalis, Streptococcus pyogenes* (less common)
   b. *Staphylococcus aureus*, Group B streptococcus, and gram-negative enteric bacilli may be seen as pathogens in the neonatal period.
4. Clinical presentation
   a. Represents a continuum of an illness that begins as an asymptomatic otitis media with effusion to acute otitis media with effusion plus signs and symptoms of an infection
   b. History varies from irritability, poor feeding, and ear-pulling in infants, to otalgia and hearing loss in older children. Upper respiratory symptoms are often present.
   c. Physical examination
      (1) Cerumen may obscure visualization and prevent initial diagnosis; if present, it should be removed.
      (2) The tympanic membrane is usually red or cloudy and may be full or bulging (bony landmarks are difficult to discern). Otorrhea may be present if spontaneous perforation has occurred. However, the most reliable sign of acute otitis media is decreased mobility of the tympanic membrane on insufflation.
      (3) Fever is present in only one-third of patients.
5. Management
   a. "Wait and see prescription" for treatment of acute otitis media in children >2 years old, unilateral involvement, and no otorrhea is becoming the standard of care. Parents are asked not to fill the antibiotic prescription for 72 hours and only if the child is no better or worsening. This approach has resulted in reduced antibiotic use in children.
   b. First-line antibiotics
      (1) Amoxicillin 80–90 mg/kg/day or cefdinir 14 mg/kg/day; despite increasing resistance, they are still considered initial agents of choice by most.
      (2) Azithromycin and clarithromycin are alternative agents for penicillin-allergic patients.
   c. Second-line agents are usually reserved for treatment failures or for patients having received antibiotics within the previous 30 days. A broad-spectrum antibiotic that provides coverage of ß-lactamase–producing species should be selected; choose one of the following:
      (1) Amoxicillin-clavulanate
      (2) Ceftriaxone (with possible repeat dosing depending on response)

d. **Patients should be advised to return in 48–72 hours if signs and symptoms do not improve and immediately if temperature increases, irritability worsens, or lethargy develops.**

e. **The possibility of systemic infection should be considered in infants with acute otitis media (especially those <2 months old) who have a fever or appear toxic. These infants require a septic evaluation and are often admitted for parenteral antibiotic therapy. In infants <2 months old, broad-spectrum coverage is recommended, because incidence of coliforms, Group B streptococcus, and *S aureus* is higher in this age group; use ampicillin plus gentamicin or ampicillin plus cefotaxime.**

E. Bullous myringitis

1. **Clinical presentation: an acute otitis media with clear or hemorrhagic blisters of the tympanic membrane associated with ear pain and mild hearing loss**
2. **Etiology: viral or bacterial (same organisms that cause acute otitis media), may also be caused by *Mycoplasma pneumoniae***
3. **Treatment: analgesics and antibiotics (same choices as for acute otitis media)**

III. MASTOIDITIS

A. Definition and etiology

1. A suppurative infection of the mastoid air cells
   a. Because the mastoid is an extension of the middle ear cleft, many patients with acute otitis media have an effusion in the mastoid. In most cases, this mastoid effusion is not considered a separate disease process.
   b. Some patients develop osteitis of the mastoid (bony destruction), which is referred to as acute surgical mastoiditis. This is what is classically referred to as mastoiditis.
2. Usually a complication of untreated or inadequately treated acute otitis media (especially if recurrent)
3. *Streptococcus pneumoniae* (most frequent isolate), *Haemophilus influenzae*, *Streptococcus pyogenes*, and *Staphylococcus aureus*

B. Clinical presentation

1. Otalgia/otorrhea and fever
2. Headache/hearing loss
3. Outward and downward displacement of the pinna
4. Posterior auricular (mastoid) tenderness, inflammation, and erythema
5. Tympanic membrane may show signs of recurrent infection (scarring, erythema, perforation)

C. Potential complications of untreated mastoiditis

1. Osteitis
2. Labyrinthitis
3. Meningitis
4. Encephalitis
5. Brain abscess
6. Damage to facial nerve (cranial nerve VII)

D. Diagnostic evaluation

1. CT scan of the temporal bone confirms diagnosis and identifies presence of complications.
2. MRI may be considered, especially if intracranial complications are suspected.

E. Management

1. Analgesia
2. Parenteral antibiotics: vancomycin (initial choice until cultures available); also consider *Pseudomonas* coverage for recurrent infection
3. Immediate ENT consult regarding the possible need for surgical debridement, which may be necessary because of poor bone penetration of antibiotics
4. Hospitalization

IV. SUDDEN HEARING LOSS

A. Pathophysiology

1. **Acute hearing loss may be associated with lesions of the external, middle, or inner ear.**
2. **Lesions of the external auditory canal, tympanic membrane, middle ear, and ossicles result in conductive hearing loss, whereas lesions of the cochlea, the auditory nerve, and brainstem auditory pathways result in sensorineural hearing loss.**

B. Etiology

1. **Conductive loss**
   a. **Otitis externa**
   b. **Impacted cerumen or foreign body**
   c. **Tympanic membrane perforation**
   d. **Sclerosis of the tympanic membrane or ossicles**
   e. **Middle ear effusion (otitis media, barotitis media)**
2. **Sensorineural loss**
   a. **Unilateral**
      (1) **Viral neuritis of the cochlear branch of cranial nerve VII**
      (2) **Acoustic neuroma**
      (3) **Meniere disease**
      (4) **Temporal bone fracture**
      (5) **Sudden sensorineural hearing loss: idiopathic hearing loss that develops suddenly or over 72 hours**
   b. **Bilateral**
      (1) **Ototoxic drug exposure**
         (a) **Antibiotics (aminoglycosides, erythromycin, vancomycin, antimalarials)**
         (b) **NSAIDs, ASA**
         (c) **Loop diuretics (furosemide, ethacrynic acid)**
         (d) **Chemotherapy**
      (2) **Exposure to loud noise**

C. Clinical presentation

1. Physical examination should include a thorough HEENT examination, including the cranial nerves; these patients frequently have associated deficits of adjacent cranial nerves, particularly V (corneal reflex) and VII as well as tinnitus, vertigo, and dysequilibrium.
2. Otoscopic examination may help distinguish patients with conductive hearing loss (due to cerumen impaction, tympanic membrane perforation, or the presence of middle ear fluid) from those with sensorineural hearing loss.
3. The Rinne and Weber tests are also useful in distinguishing conductive from sensorineural hearing loss.
   a. Rinne test: compares air conduction (AC) versus bone conduction (BC) and is performed by placing the base of a vibrating 512-Hz tuning fork against the mastoid process. When the patient can no longer hear the vibration, the tuning fork tips are placed adjacent to the external auditory canal of the ear. In patients with normal hearing, the vibrations of the tuning fork will still be heard (AC>BC).
      (1) If there is unilateral conductive loss, vibrations will not be heard (BC>AC).
      (2) If there is a unilateral sensorineural loss, test will be normal (AC>BC).
   b. Weber test: performed by placing the base of the vibrating tuning fork against the middle of the forehead and asking the patient in which ear the sound is loudest. Normally, it is equal in both ears.
      (1) If a unilateral conductive hearing loss is present, vibrations will be heard better in the ear with conductive loss.
      (2) If a unilateral sensorineural hearing loss is present, vibration will be heard better in the normal ear.
   c. If bilateral sensorineural hearing loss is present, both the Rinne and Weber tests will be normal despite a bilateral decrease in hearing acuity.

D. Diagnostic evaluation

1. CT of the temporal bone should be obtained if fracture is suspected.
2. MRI should be considered if a tumor is suspected based on the history and examination.

E. Management

1. Cerumen and foreign body impactions should be removed, and infections (otitis media or externa) treated.
2. Patients with tumors require appropriate ENT or neurosurgical consult.
3. For other patients with sudden sensorineural hearing loss, consult with an otolaryngologist for possible steroid therapy and follow-up (in 1–2 days) should be arranged.
4. If an ototoxic drug is the presumed cause of hearing loss, it should be discontinued.

V. VERTIGO

A. Definition

1. A sensation of movement of oneself (subjective vertigo) or the environment (objective vertigo)
2. Most commonly described as a feeling of spinning

B. Epidemiology

1. Vertigo is either peripheral or central in origin.
2. Peripheral vertigo accounts for 85%–90% of cases and is considered less serious prognostically than central vertigo, which accounts for 10%–15% of cases.
3. Peripheral vertigo arises from diseases of the vestibular apparatus or cranial nerve VIII and is usually self-limited.
4. Central vertigo originates from diseases of the CNS (brainstem or cerebellum).

C. Peripheral vertigo

1. Abrupt onset
2. Severe intensity
3. Brief/intermittent duration
4. Worsening with change in position
5. Associated symptoms may include nausea and vomiting, diaphoresis, tinnitus, and hearing loss
6. Nystagmus that is:
   a. Fatigable
   b. Suppressed by visual fixation
   c. Unidirectional: horizontal or horizontorotary (never vertical)
   d. No associated neurologic abnormalities

D. Central vertigo

1. Insidious onset
2. Mild intensity
3. Prolonged/continuous duration
4. Little or no effect with change in position
5. Associated symptoms may include headache, diplopia, dysarthria, dysphagia, ataxia, facial numbness, and hemiparesis.
6. Nystagmus that is:
   a. Nonfatigable
   b. Not suppressed by visual fixation
   c. Multidirectional (changes direction with changes in position): horizontal, rotary, or vertical
7. Not usually associated with hearing loss

VI. TRAUMA

A. Lacerations of the auricle

1. Must be repaired carefully to avoid cosmetic defects
2. Primary goal is to reapproximate the cartilage (with absorbable sutures) and then cover the cartilage with skin.

B. Hematomas of the auricle

1. Should be drained and dressed with a compressive dressing that maintains normal ear contours; this will prevent formation of "cauliflower ear."
2. Patients should be started on oral antibiotics (eg, quinolones) and reassessed in 24 hours for reaccumulation of blood, which (if present) will require repeat drainage and dressing replacement.

C. Frostbite of the auricle

1. Should be treated by rapid rewarming
2. Use warm water (40°–42°C) irrigation.

D. Foreign body in the ear—requires urgent referral to an ENT specialist if:

1. It cannot be safely removed without damage to the tympanic membrane.
2. It is expanding organic material (eg, bean).
3. There is any chance it is a battery.

E. Perforation of the tympanic membrane

1. Pathophysiology: a penetrating object, loud noise, infection, lightning strike, or rapid changes in pressure, eg, scuba diving accidents
2. Clinical presentation
   a. Otoscopic examination usually reveals the tear. Immobility of the tympanic membrane on bulb insufflation confirms it.
   b. Acute perforations have irregular borders, sometimes with blood.
   c. Chronic perforations have smooth margins without blood.
   d. Patients with associated symptoms of complete hearing loss, nausea, vomiting, vertigo, or facial palsy need immediate ENT referral. These symptoms suggest the presence of concurrent injury to the ossicles, labyrinth, or temporal bone.
3. Management
   a. Most perforations generally heal spontaneously (especially traumatic tears), and follow-up with ENT can be on a less-urgent basis. In either case, the ear should be kept dry.
   b. If coexisting otitis media or externa is present, an otic antibiotic suspension should be prescribed.

F. Severe blunt injury to the ear

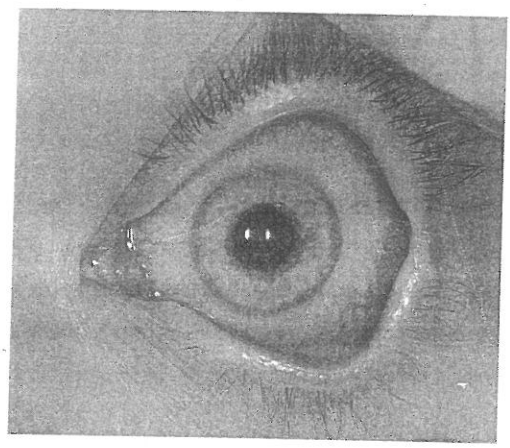
1. May be associated with fracture of the temporal bone
2. May cause hearing loss

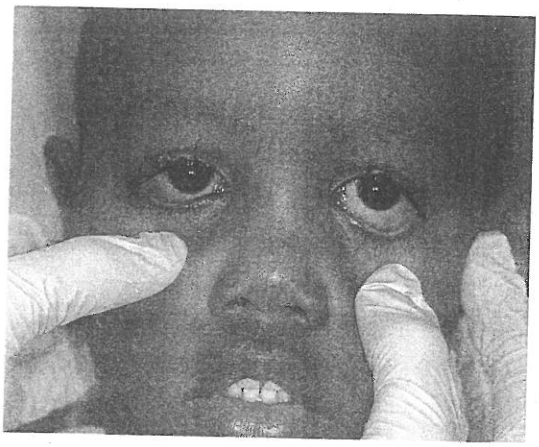
EYE EMERGENCIES

I. CONJUNCTIVITIS

A. Viral conjunctivitis ("pink eye")

1. Viruses, particularly adenoviruses, are the most frequent cause of conjunctivitis.
2. The infection is often unilateral initially but usually spreads quickly (via autoinoculation) to involve both eyes.
3. Up to 50% of patients have constitutional symptoms or a viral prodromal illness consistent with a viral syndrome (fever, rhinorrhea, myalgias, and preauricular adenopathy).
4. Clinical presentation
   a. Patients classically present with redness, itching, and tearing (watery discharge).
   b. Typical examination findings include diffuse conjunctival injection, a follicular response on the palpebral (lid) conjunctiva, a thin watery discharge, and preauricular adenopathy. (Herpes simplex keratitis and chlamydial conjunctivitis can also be associated with follicular conjunctivitis and preauricular adenopathy.)
   c. Unless an associated keratitis is present, the cornea is clear; visual acuity and pupillary reactivity are normal.





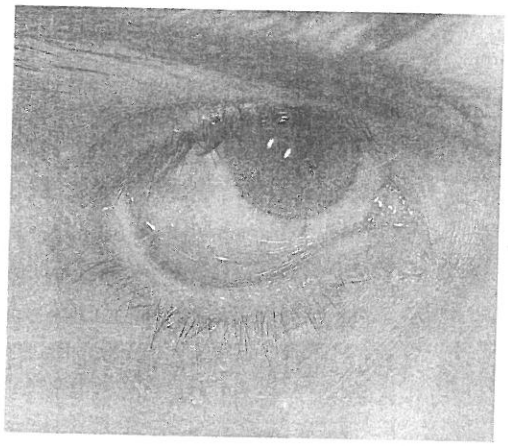
Courtesy of David Effron, MD, FACEP

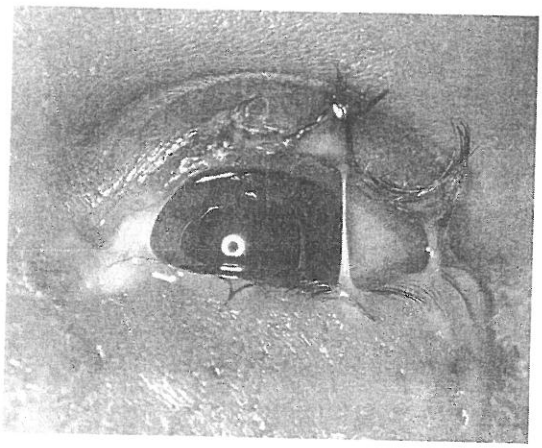
5. Infection with adenovirus types 8 and 19 (and others)
   a. Epidemic keratoconjunctivitis (a highly contagious eye infection)
   b. Clinical presentation: itchy irritated eyes → tender preauricular nodes (a few days later) → painful keratitis with severe photophobia and decreased visual acuity (1 week later)
   c. Diagnostic evaluation: fluorescein staining of the cornea reveals multiple tiny pinpoint dots.
6. Management
   a. Antibiotic therapy: because the symptoms and clinical appearance of viral conjunctivitis are usually inadequate to distinguish it from bacterial conjunctivitis, most patients are treated with a topical antibiotic.
   b. Meticulous hygiene (frequent hand washing, separate hand linens), especially with epidemic keratoconjunctivitis, because patients may be contagious for 2 weeks.

c. Cool compresses and artificial tears
d. Consider a topical vasoconstrictor–antihistamine combination for relief of symptoms.
e. Obtain prompt ophthalmologic consult for patients with evidence of keratitis.
f. Steroids should be strictly avoided.

B. Bacterial conjunctivitis

1. Clinical presentation
   a. Patients usually present with redness, a gritty sensation (like a foreign body), and discharge.
   b. The discharge is mucopurulent and often results in the eyelids being matted together on awakening.
   c. A Gram stain and culture should be obtained in all cases of neonatal conjunctivitis or whenever *Neisseria gonorrhea* conjunctivitis is suspected (see below), or with severe or worsening symptoms despite treatment.
   d. Examination findings include diffuse conjunctival injection, mucopurulent discharge, and a clear cornea that does not stain with fluorescein dye; visual acuity and pupillary responses are normal.
   e. Complications of any bacterial conjunctivitis can include corneal involvement such as keratitis, corneal ulcer formation, or even corneal perforation.
   f. Contact lens use must be discontinued until infection resolves. Topical quinolones are first-line antibiotics in contact lens wearers because of the risk of *Pseudomonas* infection.





Courtesy of David Effron, MD, FACEP

2. Types of bacterial conjunctivitis important for this examination
   a. *Staphylococcus aureus*, *Streptococcus pneumonia*, and *Haemphilus influenzae* conjunctivitis
      (1) Onset is acute.
      (2) The discharge is typically mucopurulent in nature.
      (3) White ulcers may be seen at the limbus ("marginal ulcers") as a result of an allergic reaction to the staphylococcal toxin.
      (4) There is no central corneal staining with fluorescein dye and cobalt blue light.
      (5) Management
         (a) Topical antibiotics: quinolones, aminoglycosides, or trimethoprim-polymyxin for 5–7 days

(b) Good hygiene (frequent hand washing, separate hand linens)

(c) Discontinuation of contact lens wear (Contact lens–related conjunctivitis should be treated with an aminoglycoside or quinolone to cover *Pseudomonas*.)

(d) Ophthalmologic follow-up in 2–3 days to exclude subsequent corneal involvement

b. *N gonorrhoeae* conjunctivitis (an ocular emergency)

(1) An extremely aggressive form of conjunctivitis that can ulcerate and perforate an intact cornea within hours or days

(2) Most commonly seen in newborns (ophthalmia neonatorum), typically in the first 3 days of life; also seen occasionally in sexually active adults

(3) Incubation period is 2–5 days; onset is hyperacute.

(4) Discharge is characteristically hyperpurulent and an important diagnostic clue.

(5) Diagnostic evaluation: culture (obtained with calcium alginate swab of lower conjunctiva and plated on sheep blood agar is more sensitive than Gram stain, and should be obtained in all neonatal cases and when *N gonorrhoeae* conjunctivitis is suspected)

(6) Management

(a) Parenteral antibiotic coverage for gonorrhea: IM or IV ceftriaxone

(b) Oral and topical antibiotic coverage for possible concomitant *Chlamydia*

i. Oral erythromycin (newborns and children), oral doxycycline or azithromycin (adults)

ii. Topical erythromycin

(c) Frequent ocular irrigation with a saline solution

(d) Immediate ophthalmology consult and hospital admission

(e) Evaluation and treatment of sexual partners

C. *Chlamydia* (inclusion) conjunctivitis

1. Leading cause of preventable blindness worldwide
2. Caused by an obligate intracellular parasite that produces a chronic conjunctivitis with scarring of the cornea and underside of the lids (Arlt lines)
3. Occurs in newborns (ophthalmia neonatorum), in whom an associated chlamydial pneumonia may also occur; also seen in sexually active adults
4. Clinical presentation: signs of infection occur 5–14 days after inoculation/birth (compared with only 2–5 days with gonorrhea).
5. Diagnostic evaluation: Gram stain may not be helpful (lack of organisms), but an immunofluorescent antibody screen should be positive.
6. Management

a. Systemic and topical antibiotic therapy (nearly half of infants will have concomitant chlamydial nasopharyngitis)

(1) Oral erythromycin (children and newborns); oral doxycycline or azithromycin (adults), *and*

(2) Topical erythromycin

b. Evaluation and treatment of sexual partners

D. Vernal conjunctivitis

1. One of several types of allergic conjunctivitis
2. Most commonly affects male children; a family history of atopy is common.
3. A recurrent seasonal inflammation; occurs in the warmer months of the year
4. Characterized by intense itching, burning photophobia, chemosis (edema of the bulbar conjunctiva), bilateral lid edema, cobblestone papillae under the upper lid, and a stringy mucoid discharge
5. Management
   a. A topical antihistamine preparation (eg, levocabastine) or a topical decongestant/antihistamine combination (eg, naphazoline/pheniramine)
   b. A topical mast cell stabilizer (eg, lodoxamide tromethamine or cromolyn sodium)
   c. Cool compresses
   d. If bacterial coinfection is suspected or the cause of the conjunctivitis is unclear, a topical antibiotic agent should also be prescribed.
6. A potential complication is a "shield ulcer" produced from the irritated papillae and discharge; if the ulcer becomes infected or scarred, loss of vision may occur.

E. Neonate chemical (aseptic) conjunctivitis

1. May develop a chemical conjunctivitis from topical silver nitrate prophylaxis that manifests within a few hours of delivery and resolves spontaneously within 24–36 hours
2. Diagnostic evaluation: Gram stain and culture
3. Assume a bacterial conjunctivitis if symptoms are delayed, impressive, or persistent.

II. INFLAMMATORY DISORDERS OF THE EYELIDS AND CORNEA

A. Anterior blepharitis

1. A chronic and frequently bilateral inflammation of the lid margins due to a staphylococcal infection and/or seborrheic dermatitis; itching and lid margin irritation are common symptoms.
2. Examination reveals crusty, scaly lid margins that may be erythematous and slightly swollen.



Courtesy of David Effron, MD, FACEP

3. Management
   a. Warm compresses and gentle scrubbing of the eyelids and lashes with baby shampoo (to remove debris), followed by application of a topical antibiotic ointment (erythromycin or bacitracin) to the eyelid margins daily for 4–6 weeks; warm compresses and artificial tears are also helpful.
   b. If seborrheic dermatitis of the scalp and eyebrows is present, it should be treated with selenium sulfide shampoo.
   c. Cases resistant to these measures can be treated with oral tetracycline or doxycycline for 2–3 weeks.

B. Hordeolum (stye)

1. An acute infection (usually staphylococcal) of the eyelid margin, involving either the meibomian sebaceous glands, Zeis sebaceous glands, or Moll apocrine sweat glands
2. Clinical presentation: pain, erythema, focal swelling with a tender nodule or pustule on the eyelid margin
3. Treatment: although styes often point and drain spontaneously, topical antibiotic ointments (bacitracin or erythromycin) and hot compresses hasten the process.

C. Chalazion

1. A subacute or chronic granulomatous inflammation of a meibomian sebaceous gland in the eyelid
2. Clinical presentation: a nontender bump in the mid portion of the eyelid (as opposed to the lid margin in hordeolum stye)



Courtesy of David Effron, MD, FACEP

3. Treatment
   a. Initial treatment is with a topical antibiotic ointment (bacitracin or erythromycin) and hot compresses.
   b. Persistent or chronic recurrent chalazions often respond to the addition of doxycycline for 2–3 weeks.
   c. Chalazions that persist for several weeks (or months) despite these measures may eventually require surgical incision and curettage by an ophthalmologist.

D. Dacryocystitis

1. An acute inflammation of the lacrimal sac due to infection or obstruction of the nasolacrimal duct
2. Most often seen in infants and in adults >40 years old

3. Etiology: most common causative organism is *Staphylococcus aureus*, followed by *Streptococcus epidermidis*, *Streptococcus* spp, and *Haemophilus influenzae*.
4. Clinical presentation (be able to identify on pictorial)
   a. Patients present with epiphora (overflow of tears onto the face) and an acute, unilateral, painful, red swelling below the medial canthus (location of the lacrimal sac).
   b. Digital pressure applied over this area may result in expression of purulent material from the puncta.

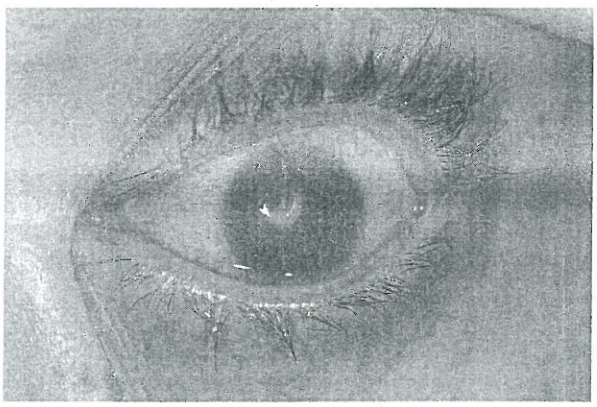


Courtesy of David Effron, MD, FACEP

5. Management
   a. Broad-spectrum oral antibiotics (eg, amoxicillin-clavulanate), topical ophthalmic antibiotics, warm compresses, gentle massage of the lacrimal sac (to empty stagnant tear accumulation), and ophthalmologic referral
   b. Signs of systemic illness may require admission.

E. Corneal ulcers (a true ocular emergency)

1. Clinical presentation
   a. Patients present with pain, foreign body sensation, photophobia, and tearing.
   b. Examination reveals conjunctival hyperemia, lid edema, and a localized whitish corneal infiltrate that has a staining epithelial defect; may also be associated with a hypopyon (accumulation of WBCs in the anterior chamber).
   c. Signs of iritis (eg, miosis, cells, and flare in the anterior chamber on slit-lamp examination) may also be present.

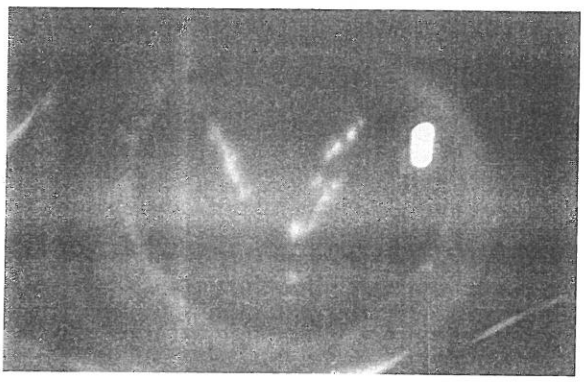


Courtesy of David Effron, MD, FACEP

2. Etiology
   a. Often seen in patients who use soft contact lenses
   b. The most common pathogen is *Pseudomonas*; *Staphylococcus* and *Streptococcus* are also common.
   c. Ulceration may lead to corneal melting and perforation. (*Pseudomonas* is particularly virulent, able to destroy the cornea in ≤24 hours).
3. Management
   a. Immediate ophthalmology consult for culture and treatment
   b. Hospital admission is rarely needed, unless the patient is a neonate or there is extensive corneal involvement.
   c. Topical broad-spectrum antibiotics (quinolones such as ciprofloxacin, gatifloxacin, moxifloxacin, or ofloxacin every 30–60 minutes) or topical fortified antibiotics (cefazolin plus gentamycin)
   d. A topical cycloplegic (cyclopentolate) for associated iritis and ciliary spasm to control pain
   e. Discontinuation of contact lens use; no patch

F. Herpes simplex keratitis

1. May be the result of primary or recurrent infection with the herpes simplex virus (usually type I).
2. Skin and mucocutaneous lesions are more common in primary infections, whereas corneal involvement (keratitis) is more common in recurrent infections.
3. Clinical presentation (be able to identify on pictorial)
   a. Patients present with ocular pain, a foreign body sensation, photophobia, and tearing.
   b. Visual acuity may be reduced.
   c. Examination reveals a diffusely reddened eye, decreased corneal sensation, and preauricular adenopathy.
4. Diagnostic evaluation
   a. Slit lamp evaluation of the unstained cornea may reveal localized corneal haziness.
   b. Fluorescein staining usually demonstrates the classic dendritic (branching) pattern over the cornea in well-established, recurrent infections; however, in primary infections and early on in recurrent infections, only a superficial punctate keratitis may be seen.



Courtesy of Dr. Thomas Steinemann
Herpes simplex virus keratitis ghost dendrite

5. Management
   a. Prompt ophthalmology consult regarding specific treatment recommendations and follow-up is mandatory; these infections are aggressive and may lead to corneal destruction resulting in permanent disability.
   b. A topical antiviral agent (eg, trifluridine or vidarabine) or oral antiviral agent (eg, acyclovir, valacyclovir, or famciclovir)
   c. A topical cycloplegic (eg, cyclopentolate or homatropine) for pain relief from ciliary muscle spasm if an associated iritis is present
   d. Although topical steroids are occasionally prescribed by ophthalmologists for patients with refractory or advanced stromal keratitis, they can promote rapid progression of the corneal epithelial infection and are contraindicated in all other forms of herpetic keratoconjunctivitis; they should not be prescribed by the emergency physician.

G. Herpes zoster ophthalmicus

1. Results from reactivation of latent varicella zoster virus in the trigeminal ganglion
2. Clinical presentation
   a. Patients present with pain, paresthesias, tearing, and a unilateral vesicular eruption in the dermatome supplied by the ophthalmic branch (V1) of the trigeminal nerve (cranial nerve V).
   b. Ocular involvement can range from conjunctivitis to iritis, keratitis, corneal anesthesia, or ocular muscle palsies; lesions on the tip of the nose (Hutchinson sign) signal nasociliary nerve involvement and a very high likelihood of ocular lesions.

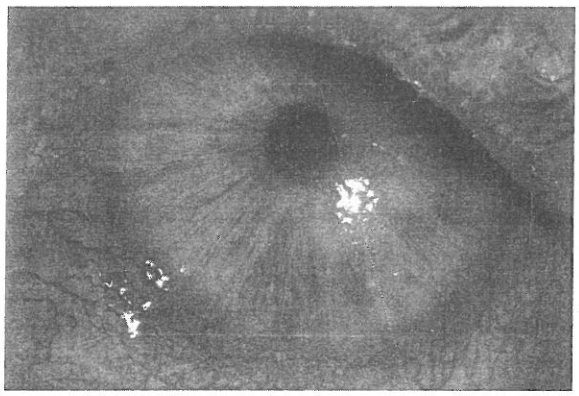


Courtesy of David Effron, MD, FACEP

3. Management
   a. Immediate ophthalmology consult
   b. Oral acyclovir, valacyclovir, or famciclovir
   c. A topical broad-spectrum antibiotic to prevent secondary infection
   d. A topical cycloplegic agent and a topical steroid (in consultation with an ophthalmologist) if iritis is present
   e. Oral analgesics as needed

H. Ultraviolet keratitis

1. Prolonged ultraviolet radiation exposure can cause damage to the corneal epithelium.
2. Typical sources of ultraviolet radiation
   a. Arc welding (welders' keratitis/flash)
   b. Reflected sunlight, especially at high altitude (snow blindness)
   c. Artificial sunlight (tanning booths, sun lamps)
3. Clinical presentation
   a. Symptoms develop 6–8 hours after exposure and include severe pain, photophobia, a foreign body sensation, tearing, and blepharospasm.
   b. There is decreased visual acuity, conjunctival injection, and a diffuse punctate keratopathy (multiple pinpoint fluorescein-stained dots on the corneal surface seen with cobalt blue light).



Courtesy of Dr. Thomas Steinemann
Severe ultraviolet keratitis

4. Management
   a. A topical cycloplegic agent (eg, cyclopentolate or homatropine) for pain control
   b. A topical broad-spectrum antibiotic ointment
   c. Oral analgesic
   d. Topical anesthetics are contraindicated in all cases of keratitis, because they can inhibit corneal healing and lead to corneal ulceration.
   e. Follow-up with ophthalmology in 24 hours

III. PERIORBITAL AND ORBITAL CELLULITIS

A. Periorbital (preseptal) cellulitis

1. An infection confined to superficial tissues that are anterior to the orbital septum (the fascial layer that acts as a partition separating the eyelids from the orbit)
2. More common and generally less serious than orbital cellulitis
3. Children (particularly those <3 years old) are most commonly affected.
4. Etiology
   a. Streptococci, staphylococci, and *Haemophilus influenzae* are the most common pathogens.

b. *H influenzae* has been more common in young children, but because of the *H influenzae* B vaccine its incidence in this population is decreasing.

5. Clinical presentation
   a. May arise by hematogenous spread (bacteremia) from otitis media or pneumonia or by direct extension from ethmoid sinusitis, skin infections, or trauma
   b. Patients present with erythema, edema, warmth, and tenderness of the eyelid, conjunctival injection, and occasionally fever.
   c. However, visual acuity, eye movements, and pupillary findings are normal (which helps distinguish periorbital from orbital cellulitis).



Courtesy of David Effron, MD, FACEP

6. Diagnostic evaluation
   a. Laboratory studies
      (1) Blood cultures
      (2) CT of the orbits (thin cuts with coronal reconstruction) if orbital cellulitis cannot be definitively excluded by clinical evaluation
   b. Soft-tissue aspirates for Gram stain and culture are no longer recommended because of risk of septum perforation with subsequent risk of deeper infection.
7. Management
   a. A broad-spectrum antibiotic that covers streptococci, staphylococci, and H influenzae; older children and adults with early/mild periorbital cellulitis can be treated on an outpatient basis with an oral antibiotic (eg, amoxicillin-clavulanate) and follow-up in 24 hours.
   b. Adults with more advanced infections and children <5 years old should be admitted for parenteral antibiotics (ceftriaxone or vancomycin) because of the increased incidence of associated bacteremia and meningitis.
   c. Ophthalmology consult

B. Orbital (postseptal) cellulitis

1. Infection of the tissues within the orbit posterior to the orbital septum. Preseptal cellulitis invariably coexists in these patients.
2. Affects all age groups but is more common in children
3. Etiology
   a. Most cases arise from direct extension of a sinus infection.

b. *Staphylococcus aureus, Streptococcus pneumoniae,* and *Haemophilus influenzae* (the bacteria associated with acute sinusitis) are the most common etiologic agents.

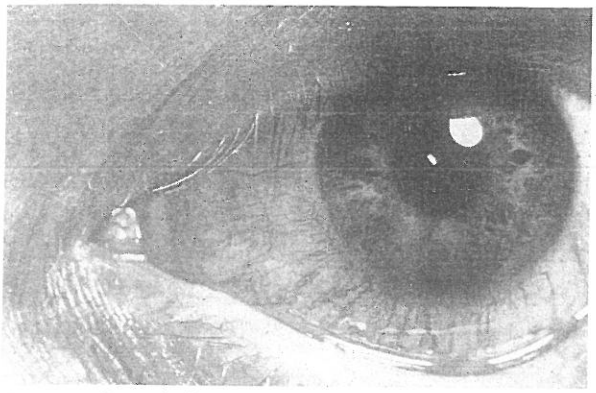
c. Mucormycosis should be considered in diabetic or immunocompromised patients.

4. Clinical presentation
   a. Patients present with the same findings seen in those with periorbital cellulitis but also have chemosis, ocular pain, pain with extraocular movements, limitation of extraocular muscle function and retropulsion with pressure, pupillary paralysis, and proptosis.
   b. Decreased visual acuity, increased intraocular pressure, and loss of sensation in the ophthalmic and maxillary branches of the trigeminal nerve may also be present.
5. Orbital compartment syndrome can occur and is suggested by afferent pupillary defect, decreased vision, significant proptosis, and significantly increased intraocular pressure. Orbital compartment syndrome is a surgical emergency and necessitates immediate ophthalmologic consult, and emergent canthotomy and cantholysis.
6. Diagnostic evaluation
   a. Blood cultures
   b. CT of the orbit and paranasal sinuses (thin section and coronal reconstruction)
   c. Measure intraocular pressure: if >20 mmHg with vision loss, surgical treatment might be necessary in addition to medications to decrease intraocular pressure.
7. Management
   a. Hospital admission
   b. Broad-spectrum IV antibiotic covering *S aureus, S pneumoniae, H influenzae,* and methicillin-resistant *S aureus*
   c. Ocular antihypertensives (eg, acetazolamide, timolol) if intraocular pressure >20 mmHg
   d. Immediate ophthalmology/ENT and infectious disease consults
   e. Canthotomy and cantholysis if strong evidence of orbital compartment syndrome
8. Complications
   a. Visual loss
   b. Cavernous sinus thrombosis
   c. CNS involvement
   d. Osteomyelitis

IV. ACUTE EYE PAIN (ASSOCIATED DECREASE IN VISUAL ACUITY)

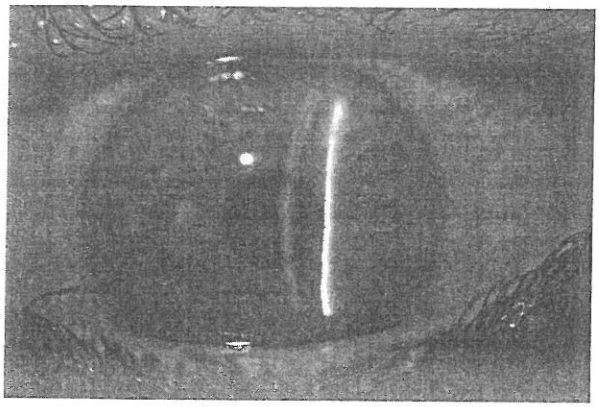
A. Acute iritis (acute anterior uveitis)

1. **Classic clinical scenario**
   a. **The patient presents with a painful red eye, severe photophobia, and blurring of vision. He or she often has a prior history of iritis or similar undiagnosed pain. Generally, only one eye is affected, and symptoms develop over time.**
   b. **Physical examination reveals a constricted and sometimes irregular pupil, a ciliary flush (diffuse reddening of the sclera at the limbus), and a decrease in visual acuity.**



Courtesy of David Effron, MD, FACEP

c. The intraocular pressure is variable; it is often decreased (due to paralysis of ciliary body function) but may be normal or increased (due to inadequate aqueous drainage). Slit-lamp examination is diagnostic and reveals flare (protein) and cells (leukocytes) in the anterior chamber. Keratitic precipitates (an accumulation of WBCs on the endothelial surface of the cornea) may also be seen.



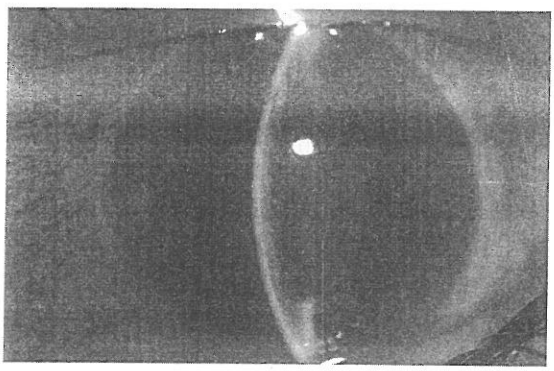
Courtesy of Dr. Thomas Steinemann
Iritis with keratitic precipitates

2. Diagnostic evaluation
   a. Presence of consensual photophobia (the photophobia produced by iritis is both direct and consensual)
   b. Presence of pain unrelieved by diagnostic application of a topical anesthetic agent; unlike the superficial pain associated with a corneal abrasion that is readily relieved by the diagnostic application of a topical anesthetic agent, the deep-seated ache of iritis is not.
3. Etiology
   a. Trauma (occurs 1–4 days after trauma)
   b. Seronegative arthritides (idiopathic ankylosing spondylitis, Reiter syndrome, etc)
   c. Inflammatory bowel disease
   d. Chronic granulomatous conditions (tuberculosis, sarcoidosis, etc)
   e. Local infection/ulcers
   f. Sexually transmitted diseases (syphilis, gonorrhea)
   g. Corneal abrasions and foreign bodies
   h. Idiopathic

4. Treatment
   a. A long-acting topical cycloplegic (eg, cyclopentolate or homatropine) to provide comfort (by eliminating ciliary spasm) and prevent formation of posterior synechiae (adhesions between the iris and lens)
   b. A topical steroid such as prednisolone acetate 1% to relieve inflammation; consult with an ophthalmologist first, and arrange follow-up in 24 hours.
5. Complications: if the pupil is not dilated, iritis may result in formation of posterior synechiae; these adhesions produce permanent pupillary disfigurement and can lead to secondary glaucoma.

B. Acute angle closure glaucoma (an ocular emergency)

1. Classic clinical scenario
   a. Occurs in patients with shallow (narrow) anterior chamber angles. Narrowing of this angle (either congenital or as a result of aging) results in closer contact between the iris and lens, producing resistance to flow of aqueous humor from the posterior to the anterior chamber (a relative pupillary block). When the angle closes completely, flow of the aqueous humor is prevented and the intraocular pressure increases abruptly.
   b. This condition is precipitated by stress or pupillary dilation by medications (parasympatholytics, sympathomimetics or anticholinergics), inadvertent administration of a topical cycloplegic in a patient with a shallow anterior chamber, or more commonly by moving from daylight to a darkened room.
   c. This is predominantly a condition of the elderly, is more common in farsighted (hyperopic) individuals and in women, and typically occurs abruptly in patients with no previous history of glaucoma.
2. Clinical presentation
   a. History: nausea/vomiting, pain (either localized to the eye/eyebrow or generalized as headache pain), blurred vision, halos around lights (due to corneal edema), and (occasionally) abdominal pain
   b. Physical examination
      (1) Decreased visual acuity
      (2) A shallow anterior chamber (causing a broad eclipse sign using an oblique flashlight)
      (3) A red, congested-looking eye with a fixed, mid-dilated, nonreactive pupil, a hazy cornea and ciliary (perilimbal) injection
      (4) Rock-hard consistency of the globe on gentle palpation
      (5) Intraocular pressure >40 mmHg (normal range 0–20 mmHg)



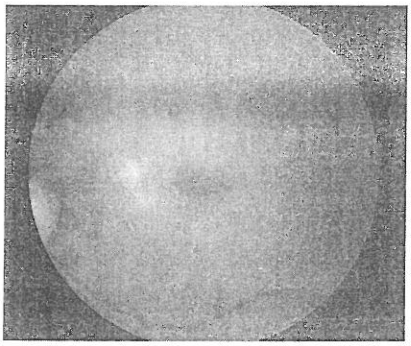
Courtesy of Dr. Thomas Steinemann

3. Management
   a. Have the patient lie completely supine (gravity may allow the lens to pull away from the iris).
   b. Pharmacologic therapy consists of the sequential administration of several different agents (which act by various means) to decrease the intraocular pressure.
      (1) Topical ß-blockers (eg, timolol), topical α-agonists (eg, apraclonidine), and oral or IV carbonic anhydrase inhibitors (acetazolamide) decrease the secretion of aqueous humor by the ciliary body.
      (2) Topical steroids (eg, prednisolone 1%) reduce inflammation.
      (3) Hyperosmotic agents (eg, mannitol) decrease the volume of fluid in the eye and rapidly lower intraocular pressure (check hourly). Note that osmotic diuretics have the greatest potential for serious adverse effects, especially in the setting of cardiovascular or renal disease.
      (4) Topical miotics (eg, pilocarpine) pull the iris back from its anterior position, thereby reopening the angle and allowing egress of aqueous humor; they do not usually have an effect until the intraocular pressure is <40 mmHg because of pressure-induced ischemic paralysis of the iris. Also treat the unaffected eye prophylactically, because the chamber angle is usually narrow in both eyes and is also at risk of acute closure.
      (5) Analgesics and antiemetics as needed
   c. Immediate ophthalmology consult
   d. Definitive treatment is surgical; bilateral laser iridotomy is performed once the acute episode resolves and the cornea clears.

V. ACUTE VISUAL LOSS

A. Central retinal artery occlusion (a true ocular emergency)

1. Classic clinical scenario
   a. The patient experiences sudden, painless, monocular loss of vision due to a thrombotic plaque or embolus (more common) of the central retinal artery.
   b. Physical examination is usually normal initially, but after 1–2 hours, reveals a dilated pupil unreactive to direct light but with an abnormal consensual response to light (Marcus-Gunn pupil). Visual acuity is markedly decreased (light perception or finger counting), and funduscopic examination reveals a pale retina with a cherry-red spot in the macular area (fovea).

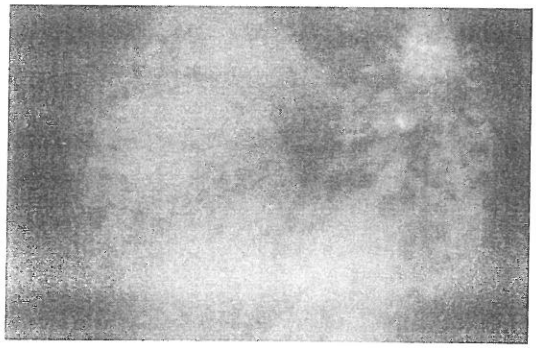


Courtesy of Dr. Thomas Steinemann

2. Patients are usually 50–70 years old and frequently have one of the following risk factors:
   a. Hypertension
   b. Carotid artery disease
   c. Diabetes mellitus
   d. Cardiac disease (atrial fibrillation, valvular disease)
   e. Vasculitis
   f. Temporal arteritis
   g. Sickle cell disease
   h. Glaucoma, intraocular hypertension, exophthalmos
3. Management
   a. Must be started as soon as the clinical diagnosis is suspected; an occlusion that persists >90 minutes usually results in permanent visual loss.
   b. Goal is to restore retinal artery blood flow by dislodging the clot, dilating the artery, and lowering intraocular pressure.
   c. Management
      (1) Intermittent digital massage of globe (5 seconds on, 5 seconds off) for 5–15 minutes
      (2) Acetazolamide 500 mg IV and a topical ß-blocker (timolol 0.5%) to reduce intraocular pressure
      (3) Immediate ophthalmology consult for paracentesis of the anterior chamber
      (4) Evaluate (and treat) for associated disease processes.

B. Central retinal vein occlusion

1. Classic clinical scenario
   a. The patient complains of a sudden, painless, monocular decrease in vision. The visual loss is usually noted on awakening and is due to thrombosis of the central retinal vein.
   b. The pupil reacts sluggishly to light, and the funduscopic examination reveals retinal hemorrhages and tortuous retinal veins ("blood and thunder fundus"). Edema of the disc may also be present. The degree of visual loss is variable but is typically less severe than that which occurs in association with central retinal artery occlusion.



Courtesy of Dr. Thomas Steinemann

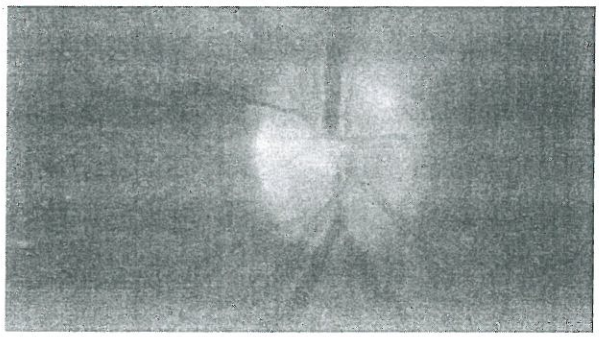
2. Risk factors
   a. Hypertension
   b. Diabetes mellitus
   c. Arteriosclerosis

d. Chronic glaucoma
e. Vasculitis

3. Management
   a. No immediate treatment is effective.
   b. Ophthalmology follow-up is required for confirmation of the diagnosis and ongoing monitoring for development of complications.
   c. Identify and treat contributing factors.
   d. Aspirin is often recommended but not proved to be beneficial.
4. Complications
   a. Neovascularization of the retina and/or iris
   b. Neovascular glaucoma

C. Optic neuritis

1. An inflammatory process and focal demyelination of the optic nerve; typically monocular in adults and binocular in children; more common in women than men
2. Characterized by reduction of central vision with preservation of peripheral vision; visual loss ranges from mild to severe (finger counting to light perception).
3. Most cases of optic neuritis are retrobulbar and do not have changes in the optic disc.
4. Classic clinical scenario: The patient is usually 15–45 years old and presents with a rapidly progressive (hours to days) reduction or blurring of vision in association with ocular pain that is worsened by eye movement.
5. Clinical presentation
   a. The presence of a Marcus-Gunn pupil (afferent pupil defect), red vision desaturation (the color red is perceived as less intense in the affected eye), and a central visual field defect confirms optic pathology.
   b. The disc may be normal (retrobulbar optic neuritis) or swollen and hyperemic (papillitis).
   c. Remainder of the eye examination is normal.



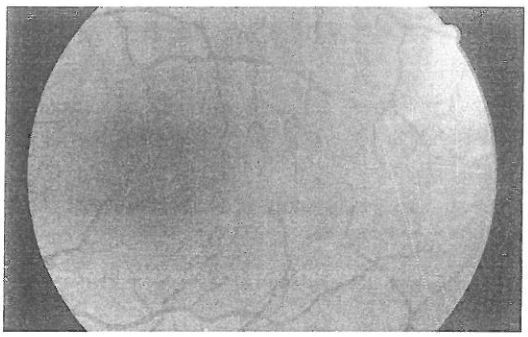
Courtesy of Dr. Thomas Steinemann

6. A clear association with multiple sclerosis exists; approximately one-third of these patients go on to develop multiple sclerosis in the ensuing years. MRI of the orbit and brain is both sensitive and specific for optic nerve inflammation and may also show demyelination in the brain.

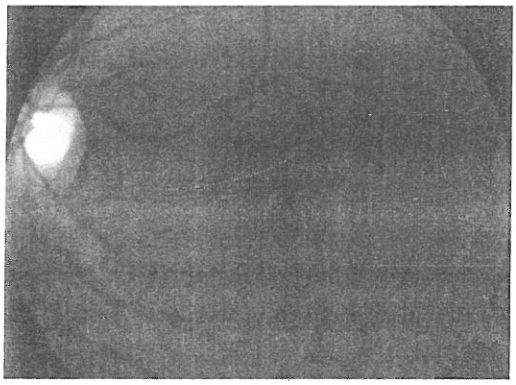
7. Management
   a. Emergent ophthalmology consult
   b. Admission for IV steroids (eg, methylprednisolone) for 3 days, followed by oral steroids for 11 days. Use of oral steroids initially is controversial and may increase the risk of recurrence of optic neuritis.

D. Eclipse burn (solar retinopathy)

1. Caused by prolonged or accidental viewing of the sun, which leads to permanent central retinal (macular) loss of vision.
2. Visual acuity is decreased, and funduscopic examination demonstrates discrete retinal disruption in the macular area.



Courtesy of Dr. Thomas Steinemann
Solar maculopathy



Courtesy of Dr. Thomas Steinemann
Solar retinopathy

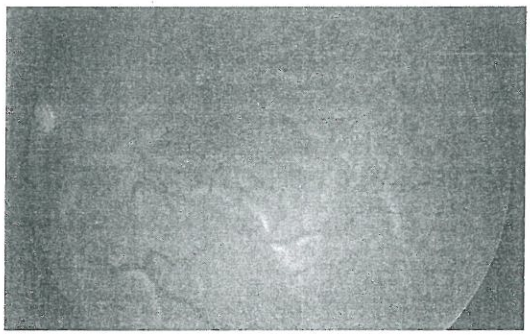
3. No treatment is effective; the patient should be referred to an ophthalmologist.

E. Retinal detachment (an ocular emergency)

1. Involves a separation of the inner neuronal retina from the outer retinal pigment epithelial layer, usually as the result of a tear in the retina from vitreous traction.
2. Painless and usually seen in patients with one or more risk factors
   a. Severe myopia
   b. Older age
   c. Trauma
   d. Previous cataract surgery
   e. An inherited connective tissue disorder (eg, Marfan syndrome)

f. Diabetes mellitus
g. A family history of retinal detachment

3. Classic clinical scenario
   a. Early symptoms are flashing lights (as the retina begins to tear) and the presence of "spider webs" or "coal dust" across the visual field (small vitreous hemorrhages). As the retina detaches, the patient experiences a sensation of a curtain that is gradually lowering or raising in front of the affected eye. Visual acuity may or may not be affected, depending on the size or involvement of the macula.
   b. Examination with a direct ophthalmoscope may reveal an undulating, dull gray, detached, or even "out of focus" retina. Direct funduscopy is not sensitive enough to exclude retinal detachment and should be suspected based on clinical symptoms. The tear itself often originates in the retinal periphery and can be seen with the indirect ophthalmoscope. Bedside ultrasound (linear probe) may be useful in the emergency department.



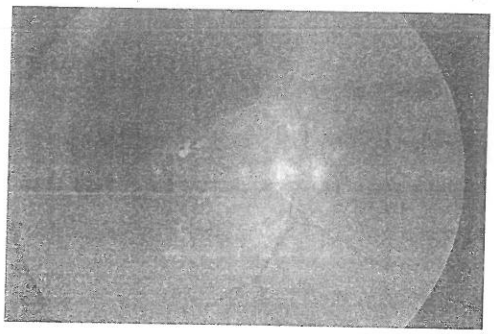
Courtesy of Dr. Thomas Steinemann

4. As long as the macula remains attached, the probability of preserving central retinal vision is very good.
5. Management
   a. Immediate ophthalmology consult for evaluation and retinal attachment surgery is indicated.
   b. The patient should avoid vigorous activity and preferably remain at bed rest until evaluated by the ophthalmologist.

F. Vitreous hemorrhage

1. A common cause of sudden, painless, unilateral loss of vision
2. Risk factors
   a. Greatest risk factor is proliferative diabetic retinopathy
   b. Posterior vitreous detachment and retinal vessel occlusion occur most commonly in older patients.
   c. Other risks include sickle cell disease, trauma, tumors, arterial microaneurysms, branch vein occlusion, and macular degeneration.
3. Classic clinical scenario
   a. If hemorrhage is preceded by retinal detachment, the patient may experience flashes of light (photopsias). Otherwise, unilateral painless vision loss occurs (often described as haze, shadows, cobwebs, or smoke).

b. On examination, the red reflex is diminished or absent (may have appearance of a red haze or a black reflex), and the fundal view is hazy. Visual acuity may or may not be affected. An afferent pupillary defect suggests a retinal detachment in addition to hemorrhage.



Courtesy of Dr. Thomas Steinemann

4. Management
   a. Immediate ophthalmology consult
   b. Bedrest with elevation of the head of the bed
   c. Some patients may benefit from surgery (photocoagulation, cryotherapy, vitrectomy).

G. Functional blindness

1. Classic clinical scenario
   a. Unilateral or bilateral blindness in the presence of normal pupillary reactions, a normal funduscopic examination, and lack of an afferent pupillary defect is very suggestive of a functional problem (hysteria/conversion disorder or malingering).
   b. An intact visual pathway can be confirmed with use of an optokinetic drum or strip, which will elicit optokinetic nystagmus.
   c. Despite the serious nature of their complaint, hysterical patients are often remarkably calm. Malingerers, on the other hand, are usually overly emotional and less cooperative.
2. Management: ophthalmologic and psychiatric referrals

H. Temporal (giant cell) arteritis

1. A vasculitis of medium and large arteries that can cause optic nerve infarction and permanent visual loss (see also page 157); it is notorious for involving the optic nerve within hours or weeks. For unknown reasons, incidence appears to be rising.
2. Classic clinical scenario
   a. Patients usually present with a unilateral headache. Sudden monocular loss of vision may also be a presenting symptom. Eye pain is not typical. Preceding symptoms may include jaw pain (claudication), weight loss, and malaise. Up to 50% of these patients have polymyalgia rheumatica (a syndrome of aching, pain, and stiffness in the proximal muscles, especially neck and shoulders); some patients may have associated neurologic symptoms that simulate a transient ischemic attack or stroke.
   b. Patients are more commonly women, and most patients are ≥50 years old.
   c. Examination can reveal scalp tenderness, temporal arteries that are tender and tortuous (and sometimes pulseless), visual loss, and an afferent pupillary defect. Funduscopic examination may be normal or show dilated retinal veins.

d. **Diagnosis is suggested by an increased sedimentation rate (>50 mm/hr). Sedimentation rate increases with age; a simple rule of thumb for estimating the upper range of "normal" for patients >50 years old is age/2 for men and (age + 10)/2 for women.**

e. **Diagnosis is confirmed by temporal artery biopsy (although treatment should not be delayed awaiting biopsy).**

3. **Management**

a. **Immediate ophthalmology consult**

b. **Administration of high-dose steroids (eg, 60 mg oral prednisone)**

VI. NONACUTE VISUAL LOSS

A. Primary open angle glaucoma

1. Most common type of glaucoma and a major cause of slowly progressive blindness in the United States
2. A painless condition of chronically increased intraocular pressure that ultimately damages the optic nerve; frequently bilateral with strong familial tendency
3. Clinical presentation
   a. Normal appearing cornea and pupil
   b. Increased intraocular pressure (>21 mmHg)
   c. Increased optic nerve cup/disc ratio (>0.4)
   d. Arcuate scotomas
   e. Gradual loss of peripheral vision with sparing of central vision
4. Management
   a. Unlike acute angle closure glaucoma, open angle glaucoma is a chronic, painless, nonemergent condition that does not generally present to the emergency department.
   b. Patients should be referred to an ophthalmologist for evaluation and treatment.
   c. Treatment: topical agents that decrease intraocular pressure by reducing the production of aqueous humor (carbonic anhydrase inhibitors, ß-blockers, and α2-agonists) and/or increasing aqueous outflow (prostaglandin agonists, sympathomimetics)

B. Cytomegalovirus (CMV) retinitis

1. A necrotizing retinitis that, if untreated, results in progressive visual loss
2. Occurs in immunocompromised patients, most notably those with advanced AIDS
3. Classic clinical scenario
   a. Although patients can be asymptomatic early on, they typically present with scintillating scotomas, visual distortions, and visual loss.
   b. Funduscopic examination reveals fluffy white retinal lesions that are usually perivascular in location and associated with hemorrhage (the characteristic "pizza pie" or "cheese and ketchup" fundus).
   c. Ophthalmic screening is recommended if the CD4 count is <50 cells/mL or with increased HIV viral load. Serum cytomegalovirus DNA testing is available for atypical cases.

4. Management
   a. Immediate ophthalmology referral and administration of valganciclovir (oral), ganciclovir (IV), or foscarnet (IV)
   b. Highly active antiretroviral therapy (HAART) for HIV infection may increase CD4 count and immune function, potentially allowing discontinuation of anticytomegalovirus therapy (which is only virustatic).

VII. TRAUMA

A. Chemical burns

1. Alkali burns (a true ocular emergency)
   a. Generally more devastating than acid burns
   b. Examples of strong alkalis include sodium hydroxide (lye), calcium hydroxide (lime in plaster and concrete), potassium hydroxide, and ammonia.
   c. Produce a liquefaction necrosis that continues to dissolve and penetrate the tissues until the alkaline substance is removed; perforation of the globe may occur.
2. Acid burns
   a. Produce coagulation necrosis of the cornea; invasion of the acid is limited by the coagulum formed.
   b. Examples of strong acids include sulfuric acid (battery acid), sodium hypochlorite (bleach), and toilet cleaners.
3. Management
   a. Immediate and copious irrigation at the scene for at least 30 minutes, followed by continuous irrigation in the emergency department using a minimum of 2 L of norma saline or lactated Ringer's per affected eye
      (1) Topical anesthetic drops should be instilled to make the patient more comfortable and tolerant of the irrigation.
      (2) Particulate matter should be removed with a moist cotton-tipped applicator.
      (3) The pH of the conjunctival fornix should be assessed periodically to monitor the progress of the irrigation:
         (a) It can be tested using nitrazine paper or a urine pH stick.
         (b) Irrigation should be continued until the pH is neutral (7.4) on two consecutiv tests 10 minutes apart; alkali burns may require several hours irrigation (up t 24 hours).
   b. Irrigation should be followed by:
      (1) A complete eye examination (including visual acuity and measurement of intraocular pressure, which requires treatment if increased)
      (2) Instillation of a topical cycloplegic (eg, cyclopentolate or homatropine) and an antibiotic ointment (eg, bacitracin, erythromycin)
      (3) Use of a nonpreserved lubricant may prevent symblepharon (adhesion of lid to conjunctiva).
      (4) Administration of oral analgesics as needed
      (5) Ophthalmology referral for follow-up in 24 hours

(6) Severe burns, particularly those due to alkalis, require immediate ophthalmology consult, discussion of additional treatment measures (eg, topical steroids), and hospital admission.

B. Lid lacerations

1. Once injury to the underlying globe has been excluded, superficial lacerations can be repaired with 6-0 or 7-0 nonabsorbable suture.
2. Lacerations in the following anatomically high-risk areas require referral for repair:
   a. Lacrimal canaliculi (lacerations of the lid margin that lie medial to the lacrimal puncta)
   b. Levator muscle/tendon (deep horizontal lacerations of the upper lid): the presence of ptosis is suggestive.
   c. Canthal tendons (penetrating wounds that transect the lateral or medial canthi)
   d. Orbital septum (deep horizontal lacerations of the upper lid): protrusion of fat into the laceration confirms septal perforation.
   e. Lid margins and lid lacerations with extensive tissue loss

C. Corneal injuries

1. Corneal abrasions
   a. Clinical presentation
      (1) Patients complain of pain, foreign body sensation, tearing, and photophobia.
      (2) Conjunctival injection is frequently present; visual acuity may be decreased if the abrasion is large or centrally located.
   b. Diagnostic evaluation
      (1) Instillation of a topical anesthetic agent provides temporary pain relief and permits a better examination. (Although topical anesthetic agents are useful diagnostically, they should *never* be prescribed for pain on an outpatient basis; corneal toxicity and retardation of epithelial regrowth occur with repetitive use.)
      (2) Always evert the lids to look for entrapped conjunctival foreign bodies; multiple fine linear abrasions of the corneal surface ("ice rink sign") are highly suggestive of a foreign body trapped under the upper lid.
      (3) Examination using a cobalt blue light source (slit lamp or Wood's light) with fluorescein staining reveals the abraded area, which appears bright green.
   c. Management
      (1) A cycloplegic agent (eg, cyclopentolate or homatropine) to relieve ciliary spasm and reduce pain
      (2) A topical antibiotic to prevent secondary infection; use an agent with gram-negative coverage (a topical quinolone [eg, ciprofloxacin, ofloxacin] or a topical aminoglycoside [eg, tobramycin, gentamicin]) for abrasions associated with soft contact lens use.
      (3) Contact lens users should be instructed to refrain from contact lens use until symptoms resolve.
      (4) Eye patching
         (a) Does not appear to facilitate healing (or provide comfort) and can have detrimental effects, especially if the injury was caused by vegetable matter or the patient wears contact lenses.
         (b) Should clearly be avoided in corneal abrasions due to prolonged contact lens use. These abrasions are associated with an increased incidence of gram-

negative bacterial infections (particularly *Pseudomonas*) and ulceration. The warm, dark, and relatively hypoxic environment created by patching may further predispose these abrasions to infection.

(5) Oral analgesics are seldom needed if the eye is adequately treated with a cycloplegic agent (eg, cyclopentolate or homoatropine; however, if the patient cannot be redosed with a cycloplegic, then oral analgesia will become necessary.

(6) Ophthalmology referral for reevaluation in 24–36 hours

2. Corneal foreign bodies and rust rings
   a. Clinical presentation: corneal foreign bodies containing iron produce a rust ring that is irritating and can be difficult to remove.
   b. Management (Note that rust ring removal is never a true eye emergency and can be deferred to a specialist if the physician does not have access to the correct equipment or is not familiar with its use.)
      (1) Apply a topical anesthetic agent, and then remove the rust ring with a commercial burr drill or a sterile 25-gauge needle under slit-lamp magnification.
      (2) The burr drill is preferred, because the removal can be accomplished more quickly and easily with this instrument.
      (3) Once the foreign body or rust ring has been removed, apply a topical broad-spectrum antibiotic (eg, erythromycin, polymixin-trimethoprim, tobramycin, ciprofloxacin, ofloxacin) and a cycloplegic agent (eg, cyclopentolate or homatropine); refer to an ophthalmologist for reevaluation in 24–48 hours.
      (4) If the rust ring cannot be easily removed, treat as above and refer to an ophthalmologist for removal the next day.
3. Corneoscleral lacerations and perforations
   a. Because of Bell phenomenon (eyeball rolling upward and outward in response to eye closure), they are commonly located in the inferior aspect of the globe.
   b. Clinical presentation
      (1) Teardrop or irregularly shaped pupil
      (2) Flattening of the anterior chamber
      (3) Small fragments of black (iris) pigment at the wound edges (uveal prolapse)
   c. Diagnostic evaluation
      (1) Examination with the cobalt blue light source of the slit-lamp with fluorescein staining will mark the area of suspected aqueous leak.
      (2) The efflux of aqueous humor will cause the fluorescein to flow from the laceration in a "riverlike" pattern (positive Seidel test), which is diagnostic if present.
   d. CT of the orbits with thin sections (cuts of 1–2 mm) and coronal reconstruction may confirm the presence of a foreign body. Bedside ocular ultrasound is *contraindicated* if globe rupture is suspected.
   e. Management
      (1) Do *not* put any pressure on the globe (not even an intraocular check with a tonopen or Schiotz tonometer).
      (2) Apply a rigid metal eye shield (*not* a patch) to protect the eye from any inward pressure.
      (3) Keep patient nothing per os (because many will need repair in the operating room).
      (4) Provide tetanus prophylaxis as needed.

(5) Administer

(a) Prophylactic IV broad-spectrum antibiotics (to cover *Staphylococcus* and *Streptococcus*) to prevent endophthalmitis

(b) Prophylactic antiemetics

(c) Analgesics as needed

(d) Update tetanus immunization

(6) Obtain immediate ophthalmology consult.

D. Ruptured globe

1. Clinical presentation
   a. Teardrop or irregularly shaped pupil
   b. Hemorrhagic chemosis
   c. Decrease in visual acuity
   d. Distorted anterior chamber
   e. Uveal prolapse
   f. Vitreous hemorrhage
   g. Positive Seidel test
2. Management: same as that for corneoscleral lacerations and punctures (see above)

E. Hyphema

1. Blunt ocular injury may cause bleeding into the anterior chamber (hyphema). The bleeding originates from the blood vessels of the ciliary body or iris.
2. Clinical presentation (be able to identify on pictorial)
   a. Although some patients with small hyphemas are asymptomatic, most complain of blurred vision, dull eye pain, and photophobia.
   b. Hyphemas are graded according to the percentage of blood in the anterior chamber.
      Grade 0: microscopic
      Grade 1: layered blood $<1/3$ of anterior chamber (most common)
      Grade 2: blood from $1/3$ to $1/2$ of anterior chamber
      Grade 3: blood from $1/2$ to less than total anterior chamber
      Grade 4: blood totally filling anterior chamber ("blackball" hyphema)
   c. Hyphemas are most easily detected when the patient is examined in the upright (sitting) position. In this position, blood will layer out in the inferior aspect of the anterior chamber, forming a meniscus.
   d. Microscopic hyphemas will go unnoticed without slit-lamp examination.
   e. A detailed eye examination should be performed to exclude associated injuries (eg, ruptured globe); the intraocular pressure should be measured, because it may be increased due to RBCs clogging the trabecular mesh work, thus impeding aqueous outflow.
3. Management
   a. Bedrest with the head of the bed elevated 45°
   b. Metal eye shield
   c. Avoidance of eye movement (eg, watching TV, reading)
   d. Analgesics as needed but no NSAIDs or ASA
   e. Antiemetics for nausea

f. Medications (ß-blocker or topical carbonic anhydrase inhibitor) to reduce intraocular pressure if >30 mmHg in a normal patient or >24 mmHg in a sickle cell patient. Avoid carbonic anhydrase inhibitors in sickle cell patients; they can cause RBCs to sickle in the anterior chamber, which clogs the trabecular meshwork and further increases intraocular pressure.

g. Immediate ophthalmology consult

h. Administration of other medications (mydriatics, cycloplegics, steroids, and antifibrinolytics) and the need for hospitalization should be decided in consultation with the ophthalmologist.

(1) A cycloplegic/mydriatic agent is often used to dilate the pupil and avoid "pupillary play" (pupil size fluctuations due to changing ambient lighting) so as not to stretch the leaking blood vessel.

(2) Steroids and antifibrinolytics may prevent rebleeding. Surgical evacuation may be necessary with large (grade IV) hyphemas.

4. Complications

a. Rebleeding-usually occurs 2–5 days after the initial bleed (when clots begin to lyse) and is the most common complication; it can cause an abrupt and sight-threatening rise in intraocular pressure.

b. Blood staining of the corneal epithelium

c. Secondary glaucoma: intraocular hypertension is seen more commonly in sickling hemoglobinopathies (sickle cell disease, thalassemia) and should be treated initially with topical ß-blockers, avoiding carbonic anhydrase inhibitors.

d. Anterior and posterior synechia formation

F. Lens subluxation and dislocation

1. Usually caused by blunt trauma to the globe but can occur spontaneously or with trivial trauma in patients with Marfan syndrome, homocystinuria, tertiary syphilis, and rheumatoid arthritis

2. Lens subluxation produces visual distortion and monocular diplopia, which persists despite covering the other eye (as opposed to binocular diplopia, which can be corrected by covering either eye).

3. Lens dislocation causes severe blurring of vision.

4. The lens may dislocate either anteriorly into the anterior chamber or posteriorly into the vitreous cavity.

a. Anterior dislocations can produce acute angle closure glaucoma by mechanically blocking the egress of aqueous humor.

b. Posterior dislocations produce iridodonesis (a quivering of the iris after rapid eye movement).

5. With subluxations, the edge of the lens will be seen on slit-lamp examination when the pupil is dilated.

6. Management

a. Obtain immediate ophthalmology consult.

b. Anterior dislocations require immediate repositioning or removal of the lens.

c. Posterior dislocations are handled more conservatively, often only with refractive correction.

G. Blowout fractures of the orbit

1. Etiology
   a. Blunt trauma to the eye transmits hydraulic forces throughout the globe. The floor and medial wall of the orbit are most susceptible to blowout fractures.
   b. Orbital floor fractures (see below) can entrap the inferior rectus and inferior oblique muscles as well as fat; contusion or laceration of the infraorbital nerve may also occur. Fractures of the medial wall of the orbit (less common; see also page 188) can entrap the medial rectus muscle.
2. Clinical presentation of orbital floor fractures
   a. Enophthalmos (posterior displacement of the eyeball causing slight ptosis)
   b. Infraorbital nerve distribution anesthesia
   c. Subcutaneous orbital emphysema (suggests a medial wall fracture with ethmoid sinus disruption)
   d. Be sure to perform a careful eye examination in these patients, because up to one in four will have concomitant injuries to the globe.
   e. Pain and diplopia on upward gaze occur secondary to limitation of upward gaze, which indicates muscular entrapment and is a true emergency.
3. Diagnostic evaluation: CT scanning is the standard of care.
4. Management
   a. Exclude entrapment.
   b. Although symptoms of fracture *without entrapment* usually resolve with conservative management, ophthalmology consult is still indicated to exclude associated injuries to the globe.
   c. Broad-spectrum prophylactic antibiotics and decongestants should be considered for those patients with sinus fractures, especially with evidence or suspicion of sinusitis. Patients with subcutaneous emphysema should be given nasal precautions (eg, no nose blowing, sneeze with mouth open, no Valsalva maneuvers).

H. Retrobulbar hematoma (an ocular emergency)

1. Etiology: extreme blunt or deep penetrating trauma to the eye produces orbital hemorrhage with accumulation of blood behind the globe.
2. Clinical presentation
   a. Exophthalmos (forward displacement of the eyeball)—may be severe
   b. Decreased visual acuity
   c. Eye pain
   d. Limited mobility of the globe
   e. Increased intraocular pressure
   f. Afferent pupil defect (or Marcus-Gunn pupil)
3. Diagnostic evaluation: CT of the orbit will demonstrate the hematoma.
4. Management
   a. Immediate ophthalmology consult for emergent decompression via a lateral canthotomy, cantholysis, and anterior chamber paracentesis.
   b. Intraocular hypertension can be treated with topical ß-blocker as well as IV carbonic anhydrase inhibitor and mannitol.

I. Intraocular foreign body

1. Clinical presentation and history
   a. Work that involves pounding metal on metal or using a grinding wheel
   b. Physical examination clue is an irregular pupil that is usually peaked toward the site of penetration.
   c. Onset of symptoms may be delayed (≥24 hours)
      (1) Reduced visual acuity
      (2) Dull, unlocalized ocular pain
2. Diagnostic evaluation
   a. Radiographic confirmation and localization of the penetrating foreign body is indicated, because these foreign bodies may need to be surgically removed.
   b. Plain radiographs of the orbit may demonstrate the foreign body.
   c. CT of the orbits (1–2 mm cuts with coronal reconstructions) are very helpful in the precise localization of intraocular foreign bodies (or associated air).
   d. MRI also provides accurate localization but is not as readily available and is *contraindicated* if potentially any metallic objects (could dislodge them).
   e. Although bedside ocular ultrasound may also identify intraocular foreign bodies, it is *contraindicated* if globe rupture is suspected.
3. Management
   a. Perform a Seidel test to see if an aqueous leak from the site of penetration is present. Do not attempt to check intraocular pressure if penetration of the globe is suspected.
   b. Place a metal shield over the eye, administer a broad-spectrum antibiotic IV, and provide tetanus prophylaxis as needed.
   c. Obtain an immediate ophthalmology consult for the decision to remove (or not) an intraocular foreign body, which is based on the composition/reactivity of the object as well as on its location in the eye.
      (1) Wood, vegetable matter, and metals (eg, iron, copper, and steel) typically incite an intense inflammatory reaction when left in the eye. BBs and pellets are also poorly tolerated, because they are composed of iron as well as lead. Emergent surgical extraction is indicated.
      (2) Inert foreign bodies (eg, glass, lead, plaster, rubber, silver, stone) that are minimally symptomatic may be treated with a nonoperative approach. However, many inert foreign bodies can cause toxicity by virtue of a coating or chemical additive, which may influence management decisions.

J. Traumatic iritis/iridocyclitis

1. Classic clinical scenario
   a. The patient has blunt ocular trauma. Onset of symptoms (aching pain, photophobia, and reduced visual acuity) usually begin 1–4 days after injury. The pupil is constricted, and a deep ciliary flush is usually present.
   b. Slit-lamp examination reveals cells and flare in the anterior chamber.
2. Management
   a. A cycloplegic agent (eg, cyclopentolate or homatropine)
   b. A topical steroid (only in consult with an ophthalmologist)
   c. Ophthalmology referral

K. Traumatic miosis and mydriasis

1. Blunt ocular trauma can produce
   a. Bruising and irritation of the iris sphincter → constriction and spasm of the pupil (miosis), while
   b. Tears in the iris sphincter muscle fibers → loss of constriction ability → dilation (mydriasis)
2. These defects may resolve spontaneously over days to weeks or, in the case of actual iris sphincter muscle fiber damage, may become permanent.
3. Management
   a. No acute treatment is indicated, unless hyphema is present.
   b. Referral to an ophthalmologist for follow-up

VIII. ANISOCORIA

A. Clinical presentation

1. Normal finding: simple physiologic anisocoria (unequal pupil size; usually ≤1 mm difference) is seen in up to 20% of the population.
2. Intracranial third nerve palsy secondary to an expanding supratentorial mass/bleed or a posterior communicating artery aneurysm (absence of an isolated third nerve palsy does not exclude an aneurysm); look for ptosis, diplopia from extraocular muscle dysfunction with "down and out" position of involved eye.
3. Prior eye surgery
4. Traumatic miosis or mydriasis
5. Uniocular drug exposure: pilocarpine 1% will constrict a pupil that has been dilated from a third nerve palsy but not from anticholinergic agent exposure.
6. Adie tonic pupil: benign condition primarily affecting women 20–40 years old who often present with blurred vision
   a. Blurred near vision but normal vision at distance
   b. Diminished deep tendon reflexes
   c. Sector palsies of iris on slit-lamp examination
   d. Slow constriction with accommodation
   e. Strong constriction to cholinergic drop (pilocarpine 0.1%)
7. Horner syndrome: due to loss of sympathetic innervation
   a. Look for ptosis, miosis, and anhidrosis
   b. "Dilatation lag" is classic, in which the involved pupil takes as much as 15 seconds to dilate, with anisocoria greatest at 5 seconds of darkness.
   c. Causes include stroke, neck mass or cancer, carotid dissection, brachial plexus trauma, and others.

B. Diagnostic evaluation

1. Compare pupil sizes in bright light and under dark conditions.

   a. With simple physiologic anisocoria, the pupil size discrepancy is the same in all levels of illumination. Furthermore, the pupils in these patients are round and respond normally to light and accommodation; associated findings (eg, ptosis) are absent.
   b. Anisocoria that is more pronounced in the dark suggests that the smaller pupil is abnormal (eg, Horner syndrome).
   c. Anisocoria that is greater in bright light suggests the larger pupil is abnormal (eg, third nerve palsy, Adie tonic pupil, or pharmacologic blockade).
2. Inquire about prior eye surgery/trauma.
3. Consider the setting and associated signs and symptoms.
   a. Head trauma and altered level of consciousness → exclude an expanding supratentorial mass/hemorrhage
   b. Headache and third nerve palsy → exclude a posterior communicating artery aneurysm
   c. Miosis, ptosis, and anhydrosis → exclude Horner syndrome
4. Ask about the use of medications.
   a. Topical parasympatholytics (eg, atropine) or sympathomimetics (eg, phenylephrine, cocaine)
   b. Nebulized anticholinergics (causing ocular exposure)
   c. Scopolamine patches (with inadvertent ocular contact via the hands)

IX. OPHTHALMIC MEDICATIONS

A. Packaging

B. Topical anesthetics

1. Used to facilitate examination of patients with painful ocular conditions and to allow performance of ocular procedures (foreign body removal, irrigation, tonometry)

Table 6: Cap/Bottle Colors of Topical Ophthalmic Medications

Medication	Cap/Bottle Color
Anesthetics	White or clear
Mydriatics and cycloplegics	Red
Miotics	Green
Antibiotics	Tan
Steroids	Pink

2. Deprive the cornea of its normal protective reflexes; in repeated doses will retard healing of the cornea
3. Should *never* be dispensed as pain medications for outpatient use
4. Types (no cross reaction between esters and amides)
   a. Esters

(1) Proparacaine 0.5%
  (a) Rapid onset
  (b) Duration of action ~15 minutes
(2) Tetracaine 0.5%
  (a) More irritating than proparacaine
  (b) Delayed onset
  (c) Duration of action ~30 minutes
(3) Fluorocaine: a premixed combination of proparacaine and fluoroscein dye that has thimerosal as a preservative (which may cause a local allergic conjunctivitis in susceptible patients)
(4) Benoxinate 0.4%
  (a) Onset 1–2 minutes
  (b) Duration of action 15 minutes

b. Amides: dibucaine 0.1%

C. Mydriatics and cycloplegics

1. Mydriatics
   a. Topical sympathomimetic agents that paralyze the iris sphincter only, thereby producing pupillary dilation (mydriasis) without affecting accommodation
   b. Used diagnostically to facilitate evaluation of internal ocular structures
   c. Example: phenylephrine 2.5%: onset 15–60 minutes, duration of action 3–4 hours
2. Cycloplegics
   a. Topical parasympatholytic agents that paralyze both the iris sphincter (mydriasis) and the ciliary muscle (cycloplegia)
   b. Used therapeutically in the treatment of iritis and deep corneal abrasions to relieve pain by eliminating ciliary spasm
   c. Use of cyclopentolate or tropicamide is preferred in the presence of acute inflammation, because their effective duration of action is shorter than that of other agents, which allows repetitive dosing to keep the patient comfortable.
      (1) Cyclopentolate: onset 30–60 minutes, duration ≤24 hours
      (2) Tropicamide: onset 15–20 minutes, duration 4–6 hours
   d. Homatropine is useful (short term) for subacute inflammation (iritis) caused by trauma or abrasion: onset 30–40 minutes, duration 2–3 days
   e. Atropine is more effective for chronic inflammation seen after surgery and in severe uveitis; because of the long duration of action (1–2 weeks), its use in the emergency department is limited.
   f. These agents are contraindicated in patients with a history of glaucoma, evidence of increased intraocular pressure, presence of a shallow anterior chamber, or suspicion of a ruptured globe.

D. Miotics

1. Pilocarpine (2%): used for treatment of acute angle closure glaucoma
2. Mechanism of action: constricts the pupil, pulling the iris back from its anterior position, which opens the angle and allows egress of the aqueous humor

E. Topical antibiotics

1. Solution (drops) are rapidly absorbed and require frequent instillation. In contrast, ointments have a longer duration of action and require less frequent administration but transiently blur vision when applied.
2. Types/examples
   a. Erythromycin: broad-spectrum, primarily bacterostatic
   b. Sulfacetamide: broad-spectrum, bacterostatic
   c. Bacitracin: broad-spectrum, primarily bacterostatic
   d. Aminoglycosides (gentamicin, tobramycin): gram-negative coverage, including *Pseudomonas*; bactericidal. Avoid neomycin-containing antibiotics because of the risk of hypersensitivity reaction in 4% of the population.
   e. Polymyxin B/trimethoprim: broad spectrum, especially gram-positive organisms such as *Staphylococcus* and methicillin-resistant *S aureus*; bacteriostatic
   f. Quinolones (ciprofloxacin, norfloxacin, ofloxacin, gatifloxacin, moxifloxacin): broad spectrum, including *Pseudomonas*; bactericidal. Drug of choice for monotherapy in treatment of corneal ulcers; poor streptococci coverage; expensive

F. Antivirals

1. Interfere with viral DNA synthesis
2. Topical antivirals (vidarabine, trifluridine)
   a. Used in treatment of herpes simplex keratitis
   b. Shorten treatment time and improve rate of healing
   c. Obtain ophthalmologic consult before prescribing.
3. Systemic antivirals: used in consultation with ophthalmology and internal medicine in the treatment of herpes simplex keratitis, herpes zoster ophthalmicus, and cytomegalovirus retinitis
   a. Acyclovir (oral and IV) is useful for both herpes simplex keratitis and herpes zoster ophthalmicus.
   b. Valcyclovir (oral) is useful for both herpes simplex keratitis and herpes zoster ophthalmicus; requires less frequent dosing than acyclovir.
   c. Famciclovir (oral) is used to treat herpes zoster ophthalmicus; requires less frequent dosing than acyclovir.
   d. Valganciclovir (oral), gangliclovir (IV), or foscarnet (IV) is used in the treatment of cytomegalovirus retinitis.

G. Topical steroids

1. Used in treatment of iritis to reduce inflammation
2. In general, they should not be prescribed in the emergency department; if they are prescribed, this should be done only in consult with an ophthalmologist, and prompt (within 24 hours) ophthalmology follow-up is indicated.
3. A primary contraindication to steroid drops is early herpes simplex keratoconjunctivitis.
4. Examples: prednisolone acetate, fluorometholone, dexamethasone

H. Hyperosmotic agents

1. Used in treatment of acute angle closure glaucoma
2. Decrease intraocular pressure by decreasing the volume of fluid in the eye

3. Examples
   a. IV mannitol (20% solution): works rapidly, very effective in the emergency department
   b. Oral glycerol (50% solution): avoid in diabetic patients because glycerol is metabolized to glucose and can produce hyperglycemia and ketosis; may cause nausea
   c. Oral isosorbide (45% solution): may cause nausea

I. Carbonic anhydrase inhibitors
   1. Used in treatment of acute angle closure glaucoma
   2. Act to reduce intraocular pressure by decreasing the secretion of aqueous humor by the ciliary body
   3. These agents are sulfa compounds; sickle cell patients who present with hyphema are at risk of red cell sickling in the anterior chamber, further compromising aqueous outflow and increasing intraocular pressure.
   4. Examples: acetazolamide (IV or oral), methazolamide (oral), dorzolamide hydrochloride (topical)

J. Topical adrenergic agents: β-blockers or α_2-agonists
   1. Used in treatment of acute angle closure glaucoma
   2. Reduce intraocular pressure by decreasing secretion of aqueous humor by the ciliary body
   3. Preferred for initial management of acute angle closure glaucoma, because they do not produce the adverse effects associated with miotics
   4. Most commonly used ß-agonist is timolol (a nonselective ß-blocker), but betaxolol is cardioselective and, therefore, preferred by some clinicians; both are packaged in bottles with yellow or blue caps.
   5. Most commonly used α_2-agonists are apraclonidine and brimonidine; they are usually packaged in bottles with white caps.
   6. All topical adrenergic agents can cause serious systemic adverse effects (especially cardiopulmonary). Topical ß-blockers can result in heart failure, heart block, and asthma, whereas α_2-agonists can cause orthostatic hypotension and syncope.

X. TONOMETRY

A. Measurement of intraocular pressure (IOP) using a calibrated device called a tonometer

B. Indicated whenever the diagnosis of glaucoma or intraocular hypertension (eg, retrobulbar hemorrhage) is considered. IOP may be low in cases of iritis.

C. Devices
   1. Schiotz impression tonometer: a handheld instrument that is available in most emergency departments and easy to use; a conversion table is required to translate the reading to mmHg.
   2. TonoPen: a handheld device that gives a digital readout of the IOP and uses disposable covers
   3. Goldman applanation tonometer: an instrument that is used in conjunction with a slit lamp
   4. Air-puff tonometer: an instrument that has the advantage of not requiring direct eye contact

D. Contraindications include the presence or suspicion of a penetrating ocular injury (absolute) or an eye infection (relative).

NASAL TRAUMA

In evaluating nasal trauma, it is important to elicit any history of previous nasal deformity or surgery.

I. NASAL FRACTURES

A. Uncomplicated nasal fracture

1. Can be determined on clinical grounds alone
2. Clinical presentation: suggestive findings include the presence of nasal bridge tenderness, swelling, ecchymosis, deformity, or crepitance. Anterior rhinoscopy may reveal septal deviation.
3. Diagnostic evaluation: radiographs add little or nothing to initial or subsequent management and need not be obtained in most isolated cases.
4. Management
   a. If patients present early (usually within 2 hours) and the swelling does not preclude an accurate assessment of nasal contour, early reduction is frequently possible.
   b. If swelling does not allow an accurate assessment, reduction can be deferred.
   c. All patients with suspected or confirmed nasal fractures should be referred to ENT for reevaluation within 2–5 days. Delaying longer may allow fractures to begin healing in their displaced position (especially in children), making intervention more difficult.

B. Complex fractures

1. Nasoethmoid fractures and nasal fractures associated with other facial fractures (eg, orbita floor fractures, tripod fractures of the zygomatic bone)
2. Diagnostic evaluation requires CT scanning to assess the extent of injury present and establish a treatment plan.
3. Management
   a. Nasoethmoid fractures are particularly complex and difficult injuries.
   b. These fractures are usually produced by a blow to the bridge of the nose and require CT scanning; possibly admission; and neurosurgical, ENT, and/or ophthalmology consults.

C. Prophylactic antibiotic therapy (antistaphylococcal)

1. Warranted in patients discharged with nasal packing and may be warranted for nasal fractures associated with a laceration of the nasal mucosa or skin (potential bacterial contamination)
2. Nasal saline irrigation helps remove blood and cleanse the mucosa; cold compresses will help reduce swelling.

II. MAJOR COMPLICATIONS

A. Septal hematoma

1. Clinical presentation: patients with nasal trauma should always be carefully evaluated to exclude the presence of a septal hematoma (a bluish purple, grapelike swelling of the nasal septum).
2. Management
   a. These subperichondral hematomas should be vertically incised and drained.
   b. After this, the anterior nasal cavity is packed to prevent blood from reaccumulating.
   c. The patient is then placed on an antistaphylococcal antibiotic and referred to ENT for follow-up in 24–48 hours.
3. Complications: if not drained, these hematomas can result in formation of a septal abscess or avascular necrosis of the nasal septum with subsequent development of a permanent "saddle-nose" deformity.

B. CSF rhinorrhea

1. Due to fracture of the cribriform plate of the ethmoid bone
2. Clinical presentation
   a. May not develop for days to weeks
   b. Should be suspected whenever a patient develops a clear nasal discharge after facial trauma and may be associated with hyposmia (or anosmia) and headache
   c. Usually unilateral and may be increased by having the patient lean forward or by compressing of the jugular vein.
3. Diagnostic evaluation
   a. The diagnosis is most reliably confirmed by high resolution CT.
   b. A positive dextrose test in the absence of epistaxis, which may cause a false-positive or a "double ring sign," is also suggestive; however, these are not sensitive, and negative results do not exclude the diagnosis.
4. Management
   a. If CSF rhinorrhea is suspected:
      (1) Place the patient in the upright position.
      (2) Collect fluid if possible.
      (3) Obtain immediate neurosurgical and/or ENT consults (depending on local practice environment).
      (4) Avoid nasal packing.
      (5) Advise patient on nasal precautions (eg, no nose blowing, sneeze with mouth open, no Valsalva maneuvers).
   b. Administration of prophylactic antibiotics (penicillin, cephalosporins) remains controversial and should be determined by the neurosurgical and/or ENT consultant.

C. Hemorrhage

1. Bleeding may be profuse but is usually of short duration.
2. If bleeding does not subside spontaneously, and CSF rhinorrhea is not suspected, nasal packing may be required; if so, be sure to prescribe an antistaphylococcal antibiotic.

NASAL FOREIGN BODIES

I. CLINICAL PRESENTATION

A. History

1. Most common in children 2–3 years old
2. History is often lacking

B. Physical examination

1. Unilateral foul-smelling nasal discharge, persistent unilateral epistaxis, or a foul body odor
2. Application of a vasoconstrictor (eg, 1% phenylephrine or oxymetazoline) and a topical anesthetic (eg, 4% lidocaine) facilitates examination (as well as subsequent treatment) by providing patient comfort and improving the visual field.

II. DIAGNOSIS AND TREATMENT

A. Inspecting the nares with a nasal speculum in combination with a head lamp usually confirms the diagnosis.

B. Removal techniques

1. Many nasal foreign bodies can be removed in the emergency department by using one of the techniques described below.
2. Method selected depends on the age and cooperativeness of the child as well as the characteristics of the foreign body.
3. Patient restraint is frequently necessary.
4. Options
   a. Positive-pressure techniques have the advantage of being simple and noninvasive and probably should be tried first. Have the child forcefully blow his or her nose while occluding the uninvolved nostril. This method works best in older, cooperative children and may require several attempts.
   b. A suction catheter with a flange may also be used to remove the foreign body.
   c. Forceps (alligator, bayonet), right angle probes, wire loops, or a small Fogarty catheter are acceptable alternatives, but particular care must be exercised to avoid injuring the mucosa or pushing the object posteriorly with these instruments.
5. After the foreign body has been removed, the nasal cavity should be inspected for the presence of additional foreign objects, infection, and trauma.
6. If the foreign body cannot be safely removed, the patient should be referred to an otolaryngologist.
   a. Follow-up on an outpatient basis in 24 hours is appropriate for most of these patients, although surgical removal is occasionally required.
   b. However, immediate ENT consult (and possible admission) are required for patients with a lodged battery, an unknown object (cannot exclude a battery), associated facial cellulitis, or associated systemic symptoms.

EPISTAXIS

I. VASCULAR SUPPLY TO THE NOSE

A. Upper half of the nose: anterior and posterior ethmoidal arteries (internal carotid → ophthalmic artery → ethmoidal arteries)

B. Lower half of the nose: sphenopalatine and greater palatine arteries (both are distal branches of the external carotid)

C. Most common sources of nosebleeds
   1. Anterior nosebleeds → anteroinferior nasal septum (Little area): Kiesselbach plexus
   2. Posterior nosebleeds, venous → posteromedial turbinate: Woodruff plexus
   3. Posterior nosebleeds, arterial → sphenopalatine artery

II. ETIOLOGY

A. Anterior bleeds (90% of all nosebleeds): more common in children (have a high level of suspicion for blood dyscrasias [eg, hemophilia, leukemia]) and young adults
   1. Trauma: look for septal hematoma
   2. Foreign body: especially children
   3. Nose picking: "epistaxis digitorum"
   4. "Winter syndrome": dry air and upper respiratory infection
   5. Allergies
   6. Nasal irritants (eg, cocaine, nasal sprays)
   7. Pregnancy: epistaxis may be due to increased blood volume and venous engorgement.
   8. Rapid changes in atmospheric pressure
   9. Infection: rhinitis, sinusitis
   10. Osler-Weber-Rendu syndrome (hereditary hemorrhagic telengectasia)

B. Posterior bleeds (10% of all nosebleeds): more common in the elderly
   1. Coagulopathy: pharmaceutical anticoagulants, blood dyscrasias, or liver disease
   2. Neoplasm
   3. Hypertension

III. CLINICAL PRESENTATION

A. History

1. Onset (spontaneous or precipitated), duration, severity
2. Isolated event or recurrent problem
3. Home management
4. Environment (eg, dry, dusty)
5. Medications and illicit drug use
6. Underlying medical problems (eg, blood dyscrasias, hepatic or renal failure, hematologic cancers)
7. Easy bruising and bleeding at other sites
8. Recent chemotherapy or prior history of neoplasm
9. Hypertension is controversial; most literature suggests no association.

B. Physical examination

1. Vital signs (note any orthostatic change)
2. Evaluate the blood and clots
3. Identify location of bleeding site
4. Signs of underlying bleeding disorder (ecchymoses, purpura, petechiae, spider angiomata)

IV. TREATMENT

A. General preparation

1. Assemble the necessary supplies and equipment at bedside; appropriately gown both the patient and yourself.
2. Then, with the patient seated and his or her head in the sniffing position, evacuate accumulated blood and clots from the nose, apply a topical anesthetic (eg, 4% lidocaine) and vasoconstrictor (eg, 1% phenylephrine or oxymetazoline), then use a nasal clamp or have the patient firmly pinch his or her nose for 10 continuous minutes.

B. Anterior sites

1. Chemical cautery (silver nitrate sticks)
2. A small piece of a hemostatic material on the bleeding site if the bleeding is mild
3. Anterior nasal packing if tamponade is needed to control the bleeding; packs are left in place for 1–3 days. Prescribe antistaphylcoccal antibiotics if packing placed.
4. Follow-up with ENT or primary care physician in 2–3 days.

C. Posterior sites

1. Posterior packing is indicated; use a gauze pack, intranasal balloon device, or Foley catheter.
2. Admit patients with posterior bleeds to an ENT specialist.
3. Closely monitor airway status, especially if history of obstructive sleep apnea.

D. Drug treatment

1. Because packing impairs sinus drainage, all patients who require anterior or posterior packing should be started on an antibiotic (eg, amoxicillin-clavulanate or a first-generation cephalosporin) as prophylaxis against sinusitis, otitis media, and toxic shock syndrome.
2. Analgesic agents and decongestants are also appropriate.
3. Aspirin and NSAIDs should be avoided.

V. COMPLICATIONS

A. May be due to the bleeding itself or as a consequence of the packs used to tamponade the bleeding

B. Severe bleeding seen more commonly with posterior bleeds; transfusion may be needed if signs of cardiovascular compromise.

C. Sinusitis and otitis media: due to obstruction of the sinus ostia and the eustachian tubes by nasal packing

D. Toxic shock syndrome: due to the rapid growth of toxin-producing staphylococcal organisms precipitated by nasal packing

E. Pressure necrosis of the columella or nasal ala: from improper padding

F. Fatal airway obstruction: precipitated by accidental dislodgment of a posterior pack into the airway

FACIAL INFECTIONS

I. SINUSITIS

A. Definition: an infection of the paranasal sinuses (ethmoid, maxillary, frontal, or sphenoid). Maxillary sinusitis is the most common. Infection of the sphenoid and frontal sinuses is least common.

1. Acute sinusitis: infection <3 weeks duration
2. Chronic sinusitis: infection >3 months duration

B. Pathophysiology

1. Results from occlusion of the sinus ostia
2. Usually precipitated by a viral upper respiratory infection or allergic rhinitis
3. Ostial obstruction → ideal culture medium in occluded sinus → secondary bacterial infection (increased likelihood after 10 days of symptoms)

C. Etiology

1. Acute sinusitis: *Haemophilus influenzae* and *Streptococcus pneumoniae* are most common; others include *Streptococcus pyogenes*, *Staphylococcus aureus*, and *Moraxella catarrhalis*.
2. Chronic sinusitis: predominantly anaerobes (>50%) or polymicrobial

D. Clinical presentation

1. Two cardinal symptoms: purulent rhinorrhea and either facial pressure or nasal obstruction; other symptoms below are also suggestive.
2. Headache or facial pain in a distribution defined by the sinuses involved
3. Swollen erythematous nasal mucosa
4. Purulent yellow-green nasal discharge
5. Low-grade fever

E. Diagnostic evaluation

1. In most instances, the diagnosis of acute sinusitis can be made based on the history.
2. Use a topical decongestant (eg, 1% phenylephrine or oxymetazoline) to better visualize purulent secretions on rhinoscopy.
3. If the diagnosis is uncertain (or complications are suspected), imaging studies may be indicated.
4. CT of the sinuses is the most sensitive technique: the "gold standard" for diagnosing sinus disease and its complications.

F. Management

1. Topical decongestants (eg, oxymetazoline or phenylephrine) for a maximum of 5 days to avoid rebound effect
2. Oral decongestants (eg, pseudoephedrine) for 2–4 weeks
3. Avoid antihistamines while acutely ill.
4. Reserve antibiotic therapy for patients with mild to moderate sinusitis who do not respond to symptomatic treatment. For those with severe or persistent symptoms (10 days) and

specific findings of acute sinusitis (nasal congestion and purulent nasal discharge), amoxicillin or TMP-SMX are favored by many as first-line agents. However, in regions with a high percentage of ß-lactamase-producing bacteria, a broad-spectrum agent should be selected: amoxicillin-clavulanate or a second- or third-generation cephalosporin (eg, cefuroxime axetil, cefprozil, cefaclor, cefpodoxime, proxetil, loracarbef). Antibiotics should be continued for 10–14 days.

5. Adjunctive therapy
   a. Analgesics
   b. Mucolytics (eg, guaifenesin)
   c. Saline irrigations
   d. Home humidifiers

G. Complications

1. Ethmoid sinusitis can lead to periorbital/orbital cellulitis and abscess formation (especially in children).
2. Frontal sinusitis may extend anteriorly and posteriorly.
   a. If it extends to the anterior table of the frontal bone, it may produce osteomyelitis of the surrounding bone ("Pott's puffy tumor"). These patients present with a doughy-feeling, tender mass above their eye.
   b. If it extends to the posterior table of the frontal bone, it may cause osteomyelitis, which can lead to:
      (1) Meningitis
      (2) Epidural abscess
      (3) Subdural empyema
      (4) Brain abscess
3. Sphenoid or ethmoid sinusitis can extend intracranially via the vascular or lymphatic channels of these sinuses, thus producing cavernous sinus thrombosis.
4. Clinical presentation
   a. Proptosis
   b. Eyelid edema
   c. Dilatation of the episcleral veins
   d. Palsies of cranial nerves III–VI
   e. Venous engorgement of the fundus
   f. Decreased mental status
   g. High fever
   h. Headache
5. Patients demonstrating these complications require admission, parenteral antibiotics, and appropriate consults.

II. PAROTITIS

A. An infection of the parotid gland that can be bacterial (usually *Staphylococcus aureus*) or viral (eg, paramyxovirus)

B. Suppurative parotitis

1. Most common in older, debilitated, or postoperative patients with decreased salivary flow due to drugs, dehydration, or irradiation
2. Etiology: *S aureus* (most common) but often polymicrobial
3. Clinical presentation
   a. Tender parotid swelling (often unilateral)
   b. Trismus
   c. Purulent discharge from the opening of Stensen (parotid) duct (opens into the mouth adjacent to the maxillary second molar)
   d. Fever
4. Management
   a. An antistaphylococcal antibiotic (eg, clarithromycin has good salivary penetration); consider *Klebsiella, Pseudomonas*, and anaerobic coverage if ill or diabetic.
   b. Hydration
   c. Local heat
   d. Massage
   e. Sialogogues (eg, sour candy, lemon juice)

C. Paramyxovirus ("mumps")

1. Epidemiology
   a. Most common in winter and spring in children 5–15 years old
   b. Rare because of childhood immunizations
2. Clinical presentation
   a. Incubation period of 12–25 days
   b. Characteristic clinical findings
      (1) Tender parotid swelling at the angle of the jaw (frequently bilateral)
      (2) Low-grade fever, headache, malaise
      (3) Clear saliva from Stensen (parotid) duct
   c. Occasionally, the infection may involve:
      (1) Gonads (epididymitis, orchitis)
      (2) Meninges (meningoencephalitis)
      (3) Pancreas (pancreatitis)
3. Management
   a. Hydration
   b. Analgesics
   c. Antipyretics
4. Infected individuals are contagious up to 6 days before to 9 days after the onset of parotid swelling.

FACIAL FRACTURES

In facial injuries, consider the possibility of concomitant cervical spine injury.

I. MANDIBULAR INJURIES

A. Hallmarks of mandibular dysfunction: limited opening or deviation on opening of the mouth, malocclusion, pain, and trismus

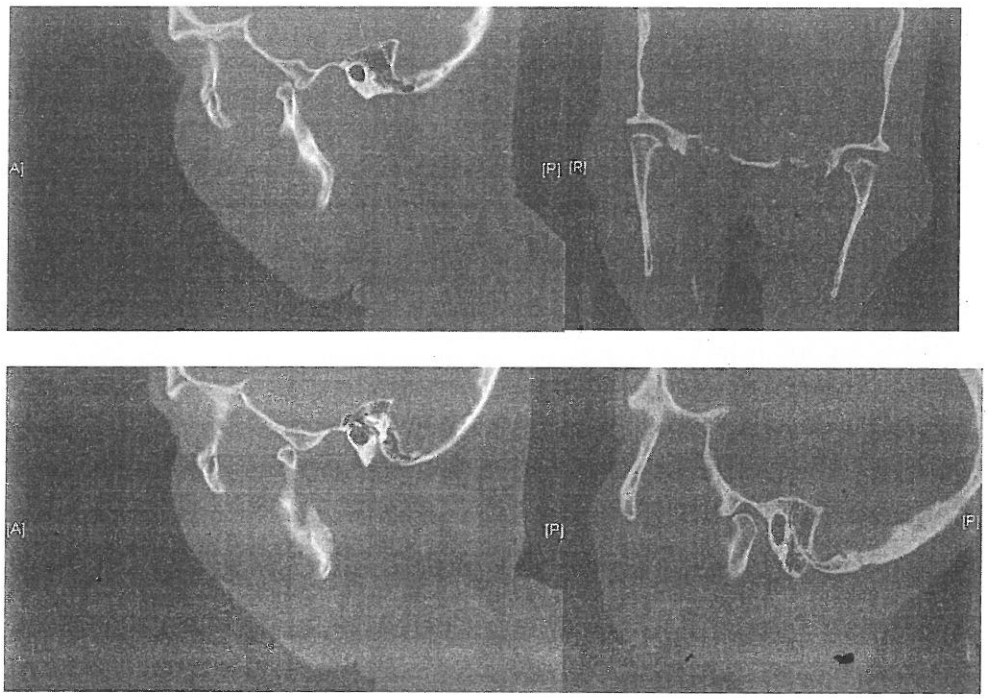
B. Mandibular fractures

1. Second most commonly fractured facial bone (nose is the first)
2. Because of its ringlike structure, the mandible fractures in two or more places in most cases (so don't stop looking after finding the first fracture).
3. The most common sites of fracture are the condyle, the body, and the angle.
4. Clinical presentation
   a. Teeth that are angulated and sometimes avulsed → alveolar fractures
   b. Lateral crossbite → unilateral condylar fractures
   c. Displacement of the lower incisors, interruption of arch continuity → symphysis fractures
   d. Ecchymosis or hematoma of the floor of the mouth → highly suspicious for a mandibular fracture
   e. Anesthesia of the lower lip → injury of the inferior alveolar or mental nerve secondary to a mandibular fracture
5. Diagnostic evaluation
   a. A dental panoramic view of the maxilla and mandible is the best study for diagnosing mandibular fractures.
   b. If a dental panoramic view cannot be done, a maxillofacial CT should be obtained.
6. Management
   a. Mandibular fractures require consult with an ENT or a plastic or oral surgeon for reduction and fixation. The time course involved (immediate or delayed) is determined by the specific injury, the presence of airway compromise, and associated injuries.
      (1) Patients with open fractures require antibiotics, and fractures should be reduced within 24 hours. Any fracture in the tooth-bearing region should be considered "open" because the periodontal ligament communicates with the oral cavity. Penicillin is the drug of choice. Clindamycin is an alternative for penicillin-allergic patients.
      (2) Patients with closed fractures that are nondisplaced may be discharged to home if there is no question of airway compromise. All subcondylar fractures and fractures in edentulous patients are considered "closed" unless accompanied by a laceration.
   b. Tetanus prophylaxis should be updated as needed.
7. Disposition
   a. If discharged, the patient should be placed on a liquid diet and given adequate analgesia; close follow-up is required.
   b. Admission is required for patients with:

(1) Airway compromise (early intubation is critical)
(2) Excessive bleeding
(3) Severely displaced fractures (impair swallowing)
(4) Grossly infected fractures
(5) Comorbid disease/disability (uncontrolled diabetes, elderly)

C. Mandibular dislocation

1. Can result from trauma, yawning, or laughing
2. The jaw is locked open (mandibular condyle is locked anterior to the articular eminence), and the patient has difficulty talking or swallowing.
3. Bilateral dislocation presents with an anterior open bite; if the dislocation is unilateral, the jaw is displaced toward the unaffected side.
4. Diagnostic evaluation
   a. Be able to identify on radiograph.
   b. Radiographic evaluation to exclude fracture should be obtained before manipulation if the dislocation is trauma related.



Courtesy of David Effron, MD, FACEP

5. Management
   a. The treatment is manual reduction with downward pressure applied to the posterior teeth to dislodge the condyle; the chin is then pressed posteriorly, so that the condyle returns to the fossa.
   b. Procedural sedation may be required if significant muscle spasm is present.
   c. After reduction, the patient should be placed on a soft diet, advised to avoid opening the mouth widely, and referred to an oral surgeon for follow-up. A muscle relaxant and an NSAID should also be prescribed.

D. Temporomandibular joint (TMJ) dysfunction

1. Clinical presentation
   a. Patients typically complain of dull, aching unilateral pain in the region of the TMJ that worsens as the day progresses. Referred ear pain is also common.
   b. On examination, there is pain on palpation of the TMJ that worsens with mouth opening and the muscles of mastication (masseter and internal pterygoid muscles). Palpable spasm of these muscles and a limited ability to open the mouth widely may also be present.
2. Management
   a. NSAIDs, a muscle relaxant, and referral to a dentist (with a specialty in TMJ disorders) for a bite splint
   b. Warm moist heat to the area, limited jaw movement, and a soft diet are also helpful.

II. MIDFACIAL FRACTURES

A. The midfacial area includes the occlusal teeth up to the lateral canthus of the eye.

B. Isolated zygomatic arch fractures

1. Clinical presentation: if depressed, the cheek may be flattened, and opening of the mouth may be painful or limited.
2. Diagnostic evaluation: these fractures are best visualized with a maxillofacial CT scan.
3. Management
   a. Treatment is generally delayed until facial swelling has resolved but should be done within 10 days.
   b. Surgical reduction is indicated for compromised mandibular excursion and unacceptable cosmesis.

C. Zygomatic-maxillary complex ("tripod") fractures

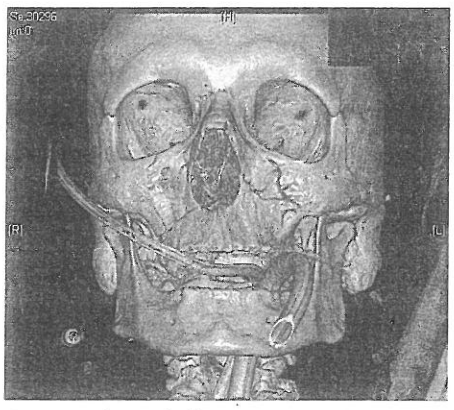
1. Generally result from a blow to the cheek and involve fractures at three sites: the zygomatic arch, zygomaticofrontal suture, and infraorbital rim. Fracture of the lateral wall of the maxillary sinus and orbital floor is also invariably present.
2. Clinical presentation
   a. A palpable step-off deformity of the inferior orbital rim
   b. Anesthesia of the cheek, upper teeth, lip, and gum from infraorbital nerve injury
   c. Periorbital swelling and ecchymosis
   d. Flattening of the cheek (if presents before significant swelling occurs)
   e. Diplopia and possible enophthalmos
3. Diagnostic evaluation: maxillofacial CT scanning is replacing plain radiographs in the evaluation of all facial fractures.
4. Management
   a. Control bleeding and arrange appropriate consult with facial trauma.
   b. Prescribe adequate analgesia.
   c. In the absence of eye involvement, patients with these fractures can often be discharged to home with follow-up in 5–7 days.
   d. Open reduction is required for open/displaced fractures.

D. Orbital floor fractures

1. May occur as isolated injuries resulting from blunt ocular trauma ("blowout" fracture) or as a component of a zygomatic-maxillary complex fracture. Orbital fat, bone, and extraocular muscles may protrude into the maxillary sinus and become entrapped. (See also blowout fractures on page 169 and in the chapter on Traumatic Disorders on pages 399–400.)
2. Clinical presentation: enophthalmos, upward gaze palsy with diplopia, hypoesthesia of the infraorbital nerve
3. Diagnostic evaluation
   a. The best view for visualizing these fractures is the modified Water view. (Be able to identify this fracture on radiograph.)
   b. CT scanning provides more detailed information.
4. Management
   a. All of these patients should be referred to ophthalmology for follow-up.
   b. Prophylactic antibiotics should be considered for patients with evidence of sinus involvement (subcutaneous emphysema, blood in the maxillary sinus).
   c. Nasal precautions
   d. Persistent enophthalmos, visual changes, and muscle entrapment are indications for surgical repair.

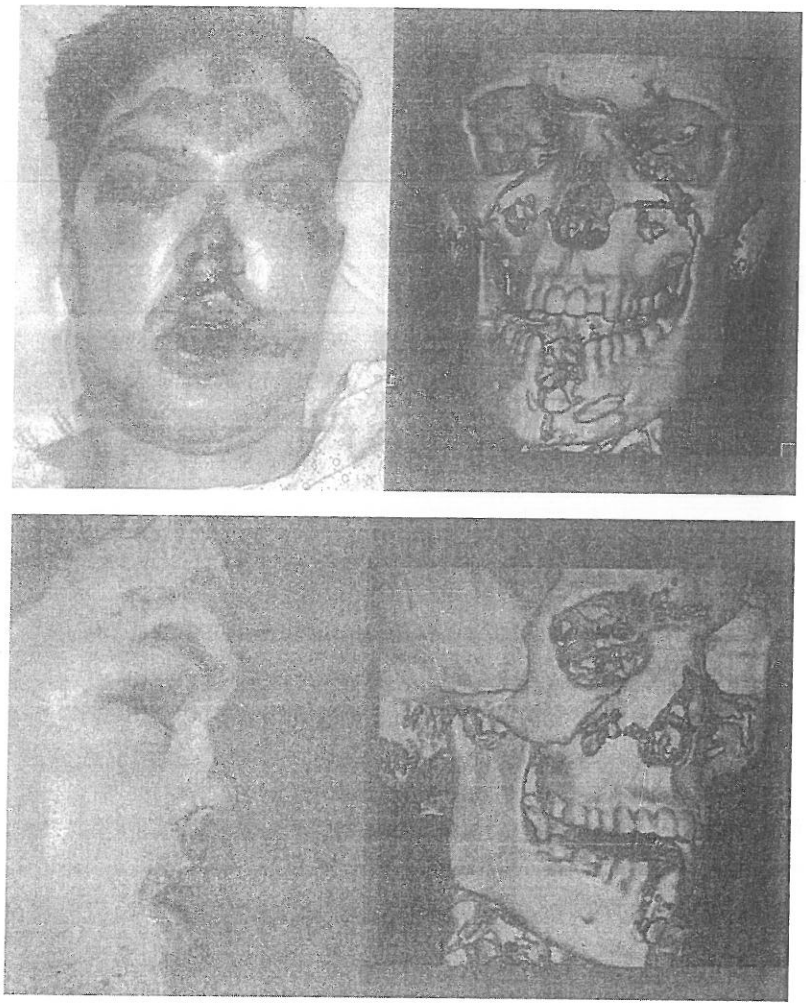
E. Maxillary fractures

1. Result from massive, direct facial trauma (eg, high-speed deceleration injuries) and may result in airway compromise
2. Classification
   a. LeFort I (palate-facial dysjunction) is a horizontal fracture of the maxilla at the level of the nasal floor.



Courtesy of David Effron, MD, FACEP

   b. LeFort II (pyramidal dysjunction) includes fractures through the maxilla, nasal bones, and infraorbital rim.
   c. LeFort III (craniofacial dysjunction) involves fractures through the zygomaticofrontal suture or zygoma and the frontal bone above the nose.



Courtesy of David Effron, MD, FACEP

3. Clinical presentation: massive soft-tissue swelling, midfacial mobility, malocclusion, and CSF rhinorrhea
4. Diagnostic evaluation: maxillofacial CT is generally needed to delineate the extent and number of fractures as well as the presence of associated injuries.
5. Management
   a. Airway management, cervical spine assessment (including associated neurologic deficits), and evaluation of the globe; vascular injury (carotid) may be associated.
   b. Administration of prophylactic antibiotics if the sinuses are involved
   c. Consult facial trauma specialist.
   d. Surgical reduction if weight-bearing buttresses involved

ACUTE AIRWAY OBSTRUCTION

I. CLINICAL PRESENTATION

A. Labored respirations (tachypnea, chest retractions, nasal flaring)

B. Stridor: a high-pitched crowing sound caused by airflow through a partially obstructed upper airway (larynx or trachea). Although it is most commonly heard during inspiration, it may be biphasic or expiratory when the obstruction is more distal (trachea).

1. Inspiratory (supraglottic or glottic): indicates obstruction above or at the larynx, eg, epiglottitis
2. Biphasic (subglottic): indicates obstruction below the larynx, eg, croup
3. Expiratory: indicates lower tracheal obstruction

C. Hoarseness

D. Dysphagia

E. Coughing

F. Cyanosis

G. Degree of airflow in the upper airway depends on the diameter of the airway in any given situation: ↓ airway diameter → ↑ resistance to airflow (by a factor of ~4).

II. DIFFERENTIAL DIAGNOSIS

A. The following conditions should be included in the differential diagnosis. Foreign body aspiration and infections are covered in more detail below.

1. Foreign body aspiration
2. Infections (croup, epiglottitis, bacterial tracheitis, retropharyngeal abscess)
3. Angioedema (hypersensitivity to irritants, allergens, ACE inhibitors)
4. Trauma (including burns, hematomas)
5. Vascular anomalies
6. Neoplasms

B. Foreign body aspiration

1. Most common in children 1–4 years old; boys are more commonly affected.
2. Although any type of object may be aspirated, peanuts are the most common offending agent and one of the most inflammatory.
3. The narrowest part of the upper airway is where most airway foreign bodies become lodged.
   a. In children → cricoid cartilage
   b. In adults → vocal cords

4. Clinical presentation
   a. The presenting signs vary with the location of the foreign body and the degree of obstruction produced.
      (1) Aphonia → complete upper airway obstruction at the level of the glottis or below
      (2) Stridor → incomplete upper airway obstruction
      (3) Wheezing → incomplete lower airway obstruction
      (4) Coughing → incomplete obstruction at the larynx or distal airways; cough is a presenting symptom in at least 80% of patients.
   b. Consider a diagnosis of foreign body aspiration in a previously well child of the appropriate age who has a history of any of the following:
      (1) A choking episode
      (2) An acute bout of paroxysmal coughing
      (3) Sudden onset of wheezing without an associated upper respiratory infection or past history of wheezing
      (4) Persistent or recurrent pneumonia
5. Diagnostic evaluation
   a. The initial approach to the child who has aspirated a foreign body (eg, a penny) is often accomplished with plain radiographs; diagnostic laryngoscopy or bronchoscopy is sometimes required, especially when the object is small and radiolucent.
   b. PA and lateral chest radiographs can be used to distinguish a radiopaque foreign body in the esophagus from one in the trachea. In the esophagus, a flat foreign body (like a coin) lies in the frontal/coronal plane and appears round in the PA view. A tracheal foreign body is generally oriented in the sagittal plane (aligning with the vocal cords) and appears round in the lateral view.
   c. The PA and lateral chest radiographs may also show indirect signs of the presence of an airway foreign body; complete bronchial obstruction produces resorption atelectasis distal to the site of obstruction, and pulmonary infiltrates may be seen in the presence of an inflammatory reaction to a foreign body (particularly vegetable matter).
   d. The presence of a radiolucent foreign body that is partially obstructing a mainstem bronchus can be demonstrated on an expiratory radiograph or bilateral decubitus chest radiographs (in the young uncooperative child). These views accentuate the "ball-valve" effect of a partially obstructing foreign body, thereby producing hyperinflation of the obstructed lung (due to air trapping) and a shift of the mediastinum away from this side. Review radiographs (expiratory and bilateral decubitus chest films) that demonstrate air trapping.
6. Management
   a. Very close airway monitoring while operating room prepared
   b. Foreign body removal in the operating room (under anesthesia) by laryngoscopy or rigid bronchoscopy

C. Infections

Croup and epiglottitis are the most important. You should know these "cold" for the exam. Look up the radiographs so that you can identify these conditions on soft-tissue radiographs.

1. Croup (laryngotracheobronchitis)
   a. Epidemiology
      (1) Age group: usually 6 months to 3 years old, with a male predominance

(2) Organism: virus (most commonly parainfluenza virus)

(3) Season: fall and winter (particularly October through December) when the causative viruses are most prevalent

(4) Site of inflammation/obstruction: glottic and subglottic area

b. Clinical presentation

(1) Prodrome: preceding viral upper respiratory infection of 2–3 days duration with a gradually increasing cough

(2) Onset and progression: insidious (over days)

(3) General appearance: nontoxic

(4) Barking cough (worse at night)

(5) Hoarse voice

(6) Respiratory distress (tachypnea, dyspnea, retractions, and stridor)

(7) Nasal discharge

(8) No or low-grade fever

c. Diagnostic evaluation

(1) In children who present with the classic clinical picture and the characteristic barking, seal-like cough, the diagnosis can be made on clinical grounds alone.

(2) When the presentation is less typical, radiographs can (when positive) confirm the diagnosis and exclude other causes of airway compromise (eg, epiglottitis, foreign body aspiration, retropharyngeal abscess). These radiographs should be taken in the emergency department as portable studies.

(3) Characteristic radiograph findings

(a) PA chest radiographs → subglottic narrowing of the tracheal air column ("steeple sign")

(b) Soft-tissue lateral of the neck → distended hypopharynx, normal epiglottis, and normal retropharyngeal space

d. Management

(1) Cool mist (use is personal preference, not evidence-based)

(2) Oxygen as needed

(3) Hydration (oral or IV)

(4) Steroids (dexamethasone 0.15-0.6 mg/kg orally or 0.6 mg/kg IM as a one-time dose) are beneficial for outpatient management.

(5) Racemic epinephrine aerosol (0.5 mL of a 2.25% solution diluted in 3 mL of saline) should be administered to patients with resting stridor or respiratory distress that is not relieved by the above measures. Although evidence to support the phenomenon of "rebound" is lacking, children sick enough to receive racemic epinephrine should be observed in the emergency department for 3–4 hours after therapy to assure that they do not return to their pretreatment stridorous state once the effect of racemic epinephrine wears off.

(6) Admission criteria

(a) Persistent stridor at rest

(b) Inability to tolerate fluids

(c) Unreliable social situation

(d) Incomplete response to racemic epinephrine

(e) Multiple doses of racemic epinephrine

(f) Severe croup at presentation (even if responsive to therapeutic measures), particularly in children <1 year old because of the smaller diameter of the airway

(g) If recurrent croup, suspect other airway abnormality.

2. Epiglottitis (supraglottitis): a true emergency

a. Epidemiology

(1) Age group: any age; 2.5 × more common in adults than children; incidence peaks in adults 20–40 years old.

(2) Organisms: since the introduction of the *Haemophilus influenzae* type B (HIB) vaccine, *Streptococcus pneumoniae* and *Staphylococcus aureus* have replaced *H influenzae* as the most common pathogens in epiglottitis in children.

(3) An association between group A ß-hemolytic streptococci and varicella-zoster virus infection leading to epiglottitis has been known for some time.

(4) Nonseasonal

(5) Site of inflammation/obstruction: supraglottic tissues, ie, the epiglottis, aryepiglottic folds, and arytenoids in children; extension to the prevertebral soft tissues, valleculae, base of the tongue, and/or soft palate may occur in adults.

b. Clinical presentation

(1) Prodrome: usually none in children; however, adults often experience a 1–2 day upper respiratory infection–like prodrome

(2) Onset and progression: typically rapid (over hours, especially if due to *H influenzae*), although adults infected with other organisms usually have a more insidious onset.

(3) General toxic appearance

(4) Patient is usually sitting in a tripod position with chin thrust forward and mouth open.

(5) Muffled ("hot potato") voice

(6) Sore throat with dysphagia and drooling

(7) Respiratory distress (tachypnea, dyspnea, and inspiratory stridor)

(8) Tachycardia out of proportion to fever, or a high fever

(9) Restlessness

(10) The diagnosis may be missed in some adults who have a more benign clinical presentation (eg, sore throat and dysphagia without evidence of acute distress) unless the possibility is considered.

c. Diagnostic evaluation

(1) Ideally confirmed in the operating room (under controlled conditions) when the swollen cherry-red epiglottis is visualized. This is particularly true in the case of patients in severe respiratory distress; they should not be disturbed for the purpose of radiographic evaluation, and attempts to visualize the epiglottis in the emergency department should be avoided.

(2) In less severe cases, diagnosis may be confirmed with a lateral soft-tissue radiograph of the neck that demonstrates the classic finding of an enlarged "thumbprint-like" epiglottis. The radiographs should be portable; if this is not feasible, someone skilled in airway management must accompany the patient to the radiography suite with airway equipment.

(3) In cooperative patients with mild to moderate symptoms and a negative lateral neck radiograph (only 40% sensitivity and 75% specificity for epiglottitis), careful direct visualization of the epiglottis may be performed to resolve the problem of false-negative results on lateral neck radiographs in patients with early epiglottitis. It is unlikely that this procedure will trigger acute airway obstruction in these patients. If direct inspection of the airway is not feasible, CT may be of value but should be done only in patients without airway distress.

d. Management

(1) Avoid agitation of the patient; allow him or her to assume a position of comfort and remain in the company of family.

(2) Provide supplemental humidified oxygen.

(3) Set up airway equipment at bedside.

(4) Obtain immediate ENT and anesthesia consults.

(5) After airway stabilization by consultants, order appropriate laboratory studies, start an IV line and fluid therapy, and begin antibiotics. Acceptable antibiotics include a second- or third-generation cephalosporin (cefuroxime, cefotaxime, ceftriaxone) or ampicillin-sulbactam.

(6) Admit to ICU.

(7) If *H influenzae* type B is isolated, household contacts require prophylaxis with rifampin, particularly if there is an unvaccinated child ≤4 years old in the contact group.

3. Bacterial tracheitis (membranous laryngotracheobronchitis): a life-threatening, bacterial infection of the subglottic region that is characterized by copious tracheal secretions. It is often superimposed on a viral upper respiratory infection and has features of both croup and epiglottitis, as well as inflammation.

a. Epidemiology

(1) Age group: 3 months to 10 years old

(2) Organism: most commonly *Staphylococcus aureus*; others include:

(a) *H influenzae*

(b) *Streptococcus pyogenes*

(c) *Moraxella catarrhalis*

(3) Nonseasonal

b. Clinical presentation

(1) Prodrome: preceding viral upper respiratory infection or croup symptoms of several days duration

(2) Onset and progress: hours to days; once bacterial superinfection occurs, progression is rapid (over hours).

(3) General toxic appearance

(4) Barking cough (present in 50% of cases)

(5) Respiratory distress (stridor and retractions)

(6) High fever

c. Diagnostic evaluation

(1) Suspect this diagnosis when a child with symptoms of viral croup becomes acutely more toxic, develops a high fever, and has progressive respiratory distress that is unresponsive to usual croup management.

(2) Direct laryngotracheobronchoscopy (performed by ENT) reveals pseudomembranes and purulent secretions and confirms the diagnosis. Removal of this debris via suctioning helps prevent airway obstruction.

d. Management

(1) Provide supplemental humidified oxygen.

(2) Obtain immediate ENT and anesthesia consults.

(3) After airway suctioning and stabilization by consultants, obtain appropriate laboratory studies (including Gram stain and culture of tracheal secretions), hydrate with IV fluids, and administer antibiotics effective against S aureus. Acceptable choices include IV nafcillin, methicillin, or oxacillin plus ceftriaxone or another third-generation cephalosporin.

(4) Admit to ICU.

ORAL AND PHARYNGEAL INFECTIONS

Infections can occur throughout the mouth and throat, and can range from minor localized infections to deep and spreading infections that may lead to airway compromise and other complications.

I. ORAL INFECTIONS

A. Masticator space abscess

1. The masticator space is the area bounded by muscles of mastication (the masseter and internal pterygoid muscles).
2. Infection is secondary, resulting from extension of an anterior space infection (buccal, sublingual, or submandibular spaces) or from an infection around the third molar.
3. Etiology: streptococci and anaerobes
4. Clinical presentation: lateral facial swelling with pain, fever, and trismus
5. Management
   a. IV antibiotics (penicillin or clindamycin are the drugs of choice)
   b. Emergent ENT or oral maxillofacial surgery consult
   c. Admission

B. Ludwig angina

1. A progressive cellulitis of the floor of the mouth; the submandibular, sublingual, and submaxillary spaces are involved bilaterally, producing massive swelling that can result in airway obstruction (33% of cases).
2. Common precipitants include an abscess of (or trauma to) the posterior mandibular molars.
3. Etiology: combination of anaerobic (*Bacteroides* spp) and aerobic (streptococci, staphylococci) oral flora
4. Clinical presentation: dysphagia, odynophagia, dysphonia, trismus, drooling, neck and sublingual pain, massive swelling/induration of the floor of the mouth and anterior neck that is brawny in character, fever, and an elevated tongue
5. Management
   a. If possible, allow the patient to maintain a sitting position; supine positioning can result in sudden airway obstruction.
   b. Set up definitive airway management equipment at bedside; the most frequent cause of death in these patients is asphyxiation.
   c. Obtain immediate ENT and anesthesia consults.
   d. Administer parenteral antibiotics (penicillin and metronidazole, cefoxitin, clindamycin, ampicillin-sulbactam, or ticarcillin/clavulanate).
   e. Admit to ICU.
6. Complications
   a. Airway compromise
   b. Extension of infection into the deeper layers of the neck or into the thoracic cavity (mediastinitis, mediastinal abscess)

II. PHARYNGEAL SPACE INFECTIONS

Review soft-tissue films of the neck with a radiologist. Ask him or her to teach you the normal size limits of the following spaces: parapharyngeal, peritonsillar, retropharyngeal, and prevertebral. Then look at radiographs that demonstrate infection or abscess formation in these spaces.

A. Parapharyngeal abscess

1. Occurs in the space lateral to the pharynx and medial to the masticator space; this space extends from the base of the skull to the hyoid bone.
2. Common precipitants of abscesses in this region are dental, pharyngeal, and tonsillar infections.
3. Etiology: combination of anaerobes and aerobes; the specific organisms isolated reflect the original site of infection.
4. Clinical presentation: neck pain, sore throat, dysphagia, odynophagia, unilateral swelling of the neck and angle of the mandible, restricted movement of the neck, torticollis, pharyngitis, bulging of the pharyngeal wall, drooling, cervical adenopathy, and fever
5. Management
   a. Emergency airway equipment set up at bedside
   b. Parenteral antibiotics (same as for Ludwig angina)
   c. Consider steroids in nonimmunocompromised hosts
   d. Emergent ENT consult
   e. Hospital admission
6. Complications: airway obstruction, spread of infection into the surrounding spaces, cranial nerve neuropathies (IX–XII), carotid artery erosion, and septic thrombosis of the internal jugular vein

B. Peritonsillar abscess

1. Occurs between the tonsillar capsule and the superior constrictor muscle; it is usually the complication of untreated or partially treated suppurative tonsillitis and is most frequently seen in teenagers and young adults.
2. Etiology
   a. Usually a polymicrobial infection caused by a mixture of aerobes and anaerobes.
   b. Group A ß-hemolytic streptococci is the predominant species. Other *Streptoccus* spp, *Haemophilus influenzae* type B, *Staphylococcus, Bacteroides,* and *Fusobacterium* spp are less frequent pathogens.
3. Clinical presentation
   a. Sore throat (≥2 days and localized to one side)
   b. Dysphagia and odynophagia → drooling
   c. Muffled ("hot potato") voice
   d. Inferior and medial displacement of the involved tonsil
   e. Soft palate fluctuance
   f. Deviation of the uvula to the opposite side
   g. Ipsilateral ear pain
   h. Trismus

i. Fever
j. Tender cervical adenopathy
k. Foul breath odor

4. Diagnostic evaluation: if you are unable to visualize the abscess, CT or ultrasound can help identify it.
5. Management
   a. IV hydration
   b. Parenteral antibiotics (penicillin, ampicillin-sulbactam, clindamycin, cefoxitin, or erythromycin)
   c. Dexamethasone
   d. ENT consult
   e. Abscess evacuation by needle aspiration or incision and drainage. While usually performed by the ENT consultant, needle aspiration may be performed by the emergency department physician if trained in the technique; incision and drainage is typically performed by ENT. Gram stain and culture of aspirated fluid should be ordered.
6. Disposition
   a. After abscess evacuation, many patients have significant relief of their symptoms and may be discharged to home on oral antibiotics (amoxicillin-clavulanate, clindamycin, or a second-generation cephalosporin). These patients should be followed up by ENT in 24 hours.
   b. If the abscess cannot be drained or the patient remains symptomatic, appears toxic, or cannot tolerate fluids, he or she should be admitted to ENT and continued on IV antibiotics.

C. Retropharyngeal abscess

1. Occurs in the space anterior to the prevertebral fascia and posterior to the pharynx; most commonly affects children 6 months to 3 years old
2. Etiology: *Staphylococcus aureus*, group A ß-hemolytic streptococci, and anaerobes are the most common pathogens.
3. Clinical presentation
   a. Sore throat
   b. Dysphagia → refusal to eat and drooling
   c. Labored respirations and (sometimes) stridor
   d. Muffled voice
   e. Fever
   f. Unilateral bulging of the posterior pharyngeal wall
   g. Swelling of the anterolateral neck with limited cervical mobility
   h. Tender anterior cervical adenopathy
   i. Systemic toxicity
   j. A preference for the supine position with the head and neck held in extension
   k. The presence of chest pain suggests that mediastinal extension has occurred.

4. Diagnostic evaluation
   a. A soft-tissue lateral radiograph of the neck is the initial study of choice. Supportive findings include widening of the retropharyngeal space with anterior displacement of the larynx and the presence of air or an air–fluid level in this space.
      (1) The normal width of the retropharyngeal space is less than half the width of the adjacent vertebral body.
      (2) The radiograph must be taken during inspiration with neck in slight extension: the expiratory phase of respiration and neck flexion → buckling and redundancy of the retropharyngeal tissues → false-positive result.
   b. The presence of chest pain should raise suspicion of mediastinal extension, and plain chest radiographs or a chest CT should be obtained.
5. Management
   a. Airway stabilization equipment at bedside
   b. IV hydration
   c. Parenteral antibiotics
      (1) A penicillinase-resistant penicillin and metronidazole *or*
      (2) Clindamycin *or*
      (3) Cefoxitin *or*
      (4) Ampicillin/sulbactum
   d. Emergent ENT consult for incision and drainage
   e. Hospital admission
6. Complications: airway obstruction, aspiration, invasion of contiguous structures (mediastinum, vessels), and sepsis

D. Prevertebral infection

1. Occurs in the space between the prevertebral fascia and the cervical spine; can be hard to differentiate from retropharyngeal infection except by patient age
2. Usually results from cervical osteomyelitis, eg, *Staphylococcus* or tuberculosis
3. Clinical presentation: may include bulging of the pharynx (usually bilateral) and tenderness of the cervical spine on palpation
4. Diagnostic evaluation
   a. Lateral neck radiographs revealing retropharyngeal swelling or osteomyelitis of the cervical spine are suggestive.
   b. CT, MRI, or a cervical myelogram are needed to confirm the diagnosis.
5. Management
   a. ICU admission
   b. Parenteral antibiotics
   c. Neurosurgical consult

PHARYNGITIS

Pharyngitis is an inflammation or infection of the mucous membranes of the oropharynx. Both noninfectious (trauma, irritant gas) and infectious (viral, bacterial, fungal) agents may b responsible. Patient complaints include dysphagia, sore throat, fever, and cervical adenopath Airway compromise is a potential complication. In the emergency department, the evaluatic of pharyngitis is primarily aimed at the diagnosis of group A β-hemolytic streptococci; appropriate treatment of this infection limits its spread, decreases the incidence of suppurati complications, prevents rheumatic fever, and hastens clinical recovery. Pharyngeal injuries, foreign bodies, and burns may present as pharyngitis.

I. TRAUMA

II. IRRITANT INHALANT

A. Many gases and vapors irritate the throat (chlorine, steam, smoke).

B. Assess respiratory effort and oxygenation; supplemental oxygen or intubation may be needed.

III. VIRUSES

Viruses are the most frequent cause of acute pharyngitis. Adenovirus, Epstein-Barr virus, influenza virus, parainfluenza virus, enteroviruses, and herpes simplex virus are commonly implicated. Cough, coryza, conjunctivitis, hoarseness, diarrhea, and stomatitis are more suggestive of viral etiology. These infections are usually self-limited and require only symptomatic treatment.

A. Infectious mononucleosis

1. Caused by the Epstein-Barr virus (human herpes virus 4); most commonly affects patient 10–25 years old
2. Clinical presentation
   a. Patients classically present with sore throat, fever, malaise, and fatigue. Abdominal p and/or left shoulder pain (Kehr sign), particularly if associated with dizziness, nausea and left upper quadrant tenderness, suggests splenic rupture (a serious complication)
   b. Examination reveals an exudative pharyngitis and tender cervical adenopathy. Poster cervical adenopathy is characteristic and helps distinguish infectious mononucleosis from other causes of pharyngitis. Hepatosplenomegaly is also common; splenic rupt may occur with minor trauma.
3. Diagnostic evaluation
   a. Peripheral blood smear reveals a lymphocytosis (>50%) and an increase in the proportion of atypical lymphocytes (>10%).

b. A monospot test (heterophile antibody) confirms the diagnosis if positive but takes 2–7 days. False-negatives may also be seen, particularly in children <4 years old.

4. Management
   a. Supportive
   b. Steroids are indicated for those who develop complications such as impending airway obstruction, severe hemolytic anemia, thrombocytopenia, or neurologic manifestations (eg, encephalitis, Guillain-Barré syndrome).
   c. Patients should also be advised to avoid contact sports for at least a month because of the potential for a traumatic splenic rupture.

B. Herpes simplex pharyngitis

1. Most commonly affects young adults and may be the result of a primary infection or reactivation
2. Characterized by the presence of grouped vesicles on an erythematous base. These lesions are exquisitely tender and subsequently erode to form superficial ulcers.
3. Treatment: antivirals (eg, acyclovir or valcyclovir) in immunocompromised patients; may also benefit immunocompetent patients but is not yet established

IV. BACTERIA

A. Group A β-hemolytic streptococci (GABHS)

1. Epidemiology
   a. Often seen in late winter
   b. Usually in patients <20 years old; rare in children <3 years old
2. Clinical presentation
   a. Patients classically have fever, tender cervical adenopathy, exudative tonsillitis, and no cough (Centor Criteria).
   b. Associated complaints of abdominal pain, vomiting, and headache are common in children.
3. Diagnostic evaluation: rapid strep screens are useful in conjunction with the Centor Criteria. Throat cultures are not routinely recommended.
4. Management
   a. Antibiotics
      (1) A 10-day course of oral penicillin VK, amoxicillin, or a single IM dose of benzathine penicillin
      (2) Azithromycin, erythromycin, or clindamycin in penicillinallergic patients
   b. Steroids
      (1) A single IM dose of dexamethasone or prednisone 60 mg orally (when given in conjunction with antibiotic therapy) decreases the duration of symptoms and provides significant pain relief.
      (2) Should be considered for patients with significant discomfort
5. Complications (both suppurative and nonsuppurative)
   a. Pharyngeal space infections (suppurative complication): appropriate antibiotic therapy of GABHS decreases the incidence of this complication.

b. Glomerulonephritis (nonsuppurative complication): treatment of GABHS is not effective in preventing this complication. Patients present after a latent period of 1.5–3 weeks with facial edema, decreased urinary output, and dark, tea-colored urine. The most useful laboratory study for making this diagnosis is the urinalysis, which reveals RBCs, WBCs, and casts. Other laboratory findings include an increased erythrocyte sedimentation rate, a mild normochromic anemia, and hyperkalemia.

c. Rheumatic fever (nonsuppurative complication): treatment of streptococcal pharyngitis within 9 days of infection prevents this complication. Patients usually present after a latent period of 2.5–5 weeks and initially complain of severe migratory joint pain. More than a third go on to develop carditis and valvular damage (mitral valve is most commonly involved). Morbidity is most closely associated with this feature. The Jones Criteria (revised), listed below, are a combination of clinical and laboratory features used to establish the diagnosis of rheumatic fever:

(1) Major
   (a) Polyarthritis
   (b) Carditis
   (c) Chorea
   (d) Erythema marginatum (nonpruritic, painless, serpiginous, erythematous rash; lesions have pale centers and dark margins; erupts on the trunk and is usually seen only in fair-skinned patients)
   (e) Subcutaneous nodules

(2) Minor
   (a) Fever
   (b) Arthalgias
   (c) History of rheumatic fever or rheumatic heart disease
   (d) Laboratory findings: leukocytosis, ↑ erythrocyte sedimentation rate, ↑ C-reactive protein, ELISA positive for GABHS, ↑ PR interval

(3) Evidence of a preceding GABHS infection (positive throat culture, ↑ antistreptolysin O titer, scarlet fever), plus the presence of 2 major (or 1 major and 2 minor criteria) make the diagnosis highly probable. Penicillin is the treatment of choice. Adjunctive therapy includes aspirin or NSAIDs for arthritis and steroids for carditis.

B. Group C and G streptococci

1. May also cause pharyngitis and can result in the same suppurative complications as GABHS
2. Linked with the development of scarlet fever and glomerulonephritis (nonsuppurative complications) but have not been shown to cause rheumatic fever
3. Infections are clinically indistinguishable from GABHS infections and should be managed in the same manner.

C. Diphtheria

1. This rare but serious cause of pharyngitis typically occurs secondary to noncompliance with diphtheria-pertussis-tetanus (DPT) immunizations. Spread is primarily by contact with respiratory secretions, and the period of incubation is ~1 week.
2. Morbidity is due to both infectious and toxic reactions.
   a. Infectious invasion causes enough tissue necrosis to produce a pseudomembrane in the posterior pharynx that can progressively enlarge and lead to airway obstruction.

b. *Corynebacterium diphtheriae* elaborates a powerful exotoxin that can cause widespread organ damage.
   (1) Myocarditis/AV block/endocarditis
   (2) Nephritis
   (3) Hepatitis
   (4) Neuritis with both bulbar and peripheral paralysis
      (a) The most commonly observed paralysis involves the intrinsic and extrinsic muscles of the eyes, leading to ptosis and strabismus.
      (b) Involvement of the palate produces a change in voice quality and difficulty speaking. (The muscles of the palate are usually the first to become paralyzed).
      (c) Limb paralysis and loss of deep tendon reflexes may also occur.

3. Clinical presentation
   a. The patient has an acute onset of sore throat, fever, and general malaise, and appears toxic and is tachycardic. His or her voice is hoarse, muffled, or even absent.
   b. Physical examination reveals an exudative pharyngitis with a white to gray, closely adherent pseudomembrane, marked cervical adenopathy ("bull neck"), and fetid breath ("dirty mouse" smell). A serosanguineous nasal discharge may also be present.
4. Diagnostic evaluation
   a. Gram stain of pharyngeal swab specimen reveals gram-positive rods with clubbing.
   b. Culture on Loeffler or tellurite media is positive.
   c. CBC may show thrombocytopenia.
5. Management
   a. ABC stabilization
   b. Definitive treatment is aimed at both the bacteria and the exotoxin and should be started as soon as the diagnosis is suspected. Do not wait for a positive culture report.
      (1) Parenteral penicillin (or erythromycin in penicillin-allergic patients) *and*
      (2) Diphtheria antitoxin
   c. All patients should be hospitalized with respiratory isolation.
   d. Recommendations for close contacts
      (1) Asymptomatic, immunized contacts should be given a tetanus-diphtheria booster if >5 years have elapsed since their last dose.
      (2) Asymptomatic, partially immunized or unimmunized contacts must receive one dose of IM penicillin (7–10 days of oral erythromycin may be used in the penicillin-allergic patient) and begin the immunization series.

D. Gonorrhea

1. Should be considered in adolescents and adults who engage in orogenital sex.
2. When it occurs in young children, sexual abuse should be suspected.
3. Treatment is with ceftriaxone (ciprofloxacin resistance is increasingly common). A regimen that is effective against *Chlamydia* (eg, azithromycin, doxycycline) should also be prescribed because of the possibility of coinfection.

V. FUNGI

A. Fungi, such as *Candida*, *Cryptococcus*, and *Histoplasma*, can produce pharyngitis in immunocompromised patients.

B. *Candida albicans* overgrowth

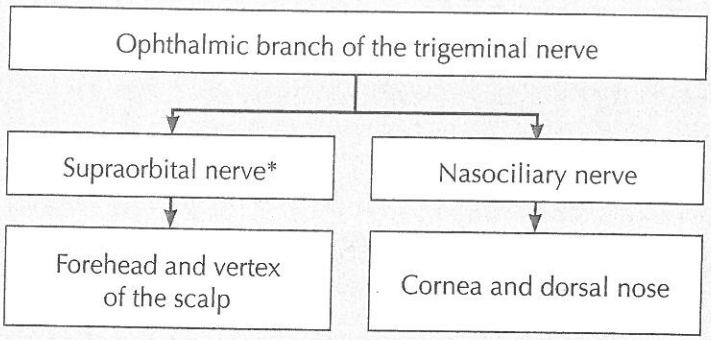
1. Most common in immunocompromised patients (eg, HIV, chemotherapy, diabetes, chror steroid use) and neonates.
2. Clinical presentation: physical examination reveals white, removable plaques on an erythematous base.
3. Treatment: nystatin swish and swallow, or systemic fluconazole

DENTAL EMERGENCIES

I. NEUROANATOMY OF THE FACE

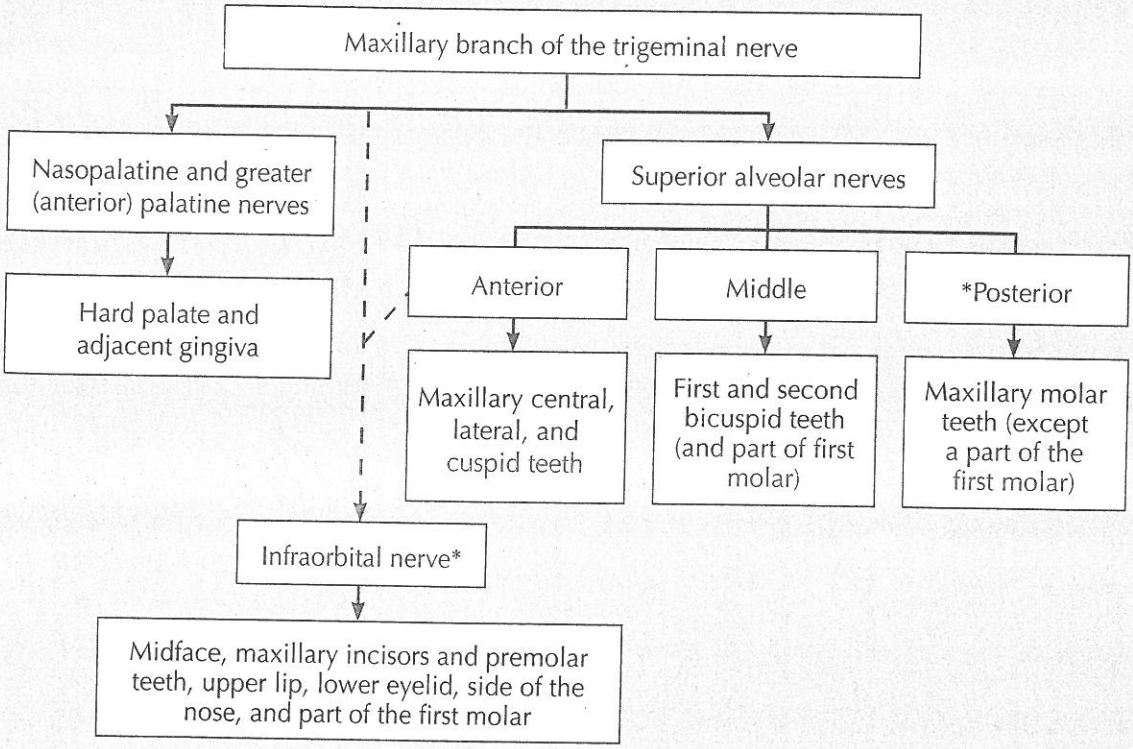
Sensory innervation of the face and mouth is via the three branches of the trigeminal nerve (cranial nerve V): ophthalmic branch, maxillary branch, and the mandibular branch. The detailed anatomy of these branches is somewhat complex, especially that of the maxillary branch. The schematics that follow have been simplified to facilitate the learning process.

A. Ophthalmic branch of the trigeminal nerve



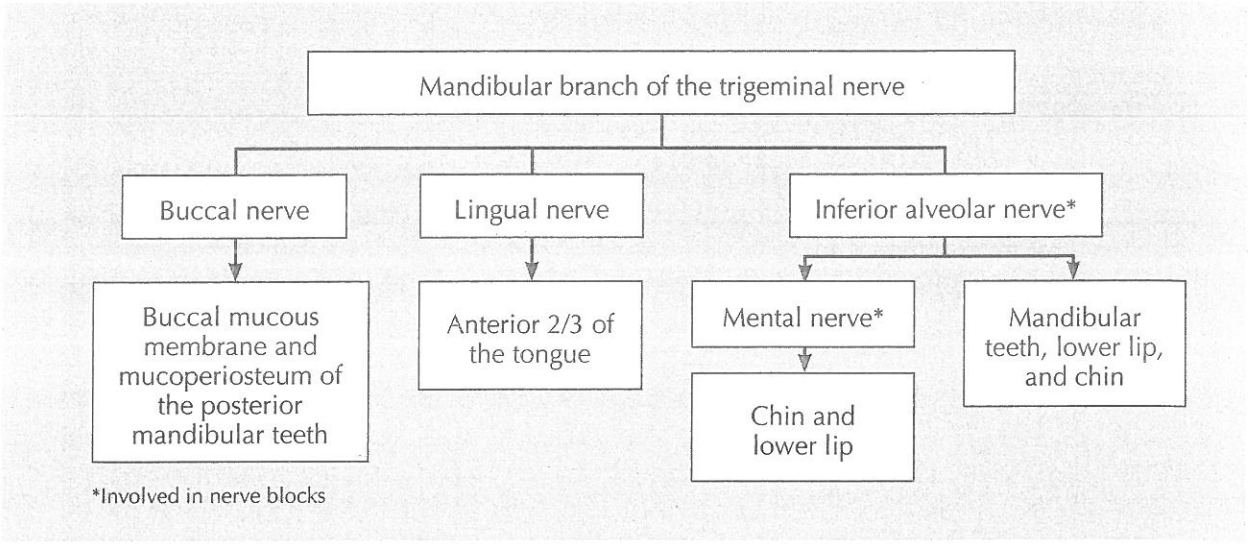
*Involved in nerve blocks

B. Maxillary branch of the trigeminal nerve



*Involved in nerve blocks

C. Mandibular branch of the trigeminal nerve



II. FACIAL AND ORAL ANESTHESIA

A. Intraoral local anesthesia can be achieved with an anesthetic agent (eg, lidocaine or bupivacaine). Unless contraindicated, a vasoconstrictor such as epinephrine (1:100,000) is generally combined with the anesthetic agent to prolong its duration of action.

B. Nerve blocks

1. Supraorbital nerve block → anesthesia to ipsilateral forehead and scalp
2. Infraorbital nerve block → anesthesia to the ipsilateral area between the lower eyelid and upper lip, including the maxillary incisors, premolars, and a portion of the first molar
3. Posterior superior alveolar nerve block → anesthesia to ipsilateral maxillary molars (except a portion of the first molar)
4. Inferior alveolar nerve block → anesthesia to ipsilateral mandibular teeth, lower lip, and chin
5. Mental nerve block → anesthesia to ipsilateral lower lip and chin; bilateral mental nerve blocks are required for midline lip lacerations due to cross-innervation.

C. Supraperiosteal infiltrations provide anesthesia to individual teeth.

D. Complications

1. Vascular injection and spasm
2. Mechanical nerve injury
3. Needle misplacement resulting in incomplete anesthesia or inadvertent anesthesia of other facial structures
4. Infections in peripharyngeal spaces (especially with inferior alveolar nerve block)

III. ANATOMY OF A TOOTH

A. Structural anatomy

1. Two main portions: crown and root
2. Enamel covers the dentin which, in turn, covers the pulp (the neurovascular supply of the tooth).
3. Embedded in the alveolar bone and held in place by cementum and a periodontal ligament.

B. Sensory innervation

1. Maxillary teeth: via the anterior, middle, and posterior superior alveolar nerves
2. Mandibular teeth: via the inferior alveolar nerve

IV. DENTAL EMERGENCIES

A. Trauma

1. Management of tooth fractures is determined by the extent of the fracture and the patient's age.
   a. Ellis I
      (1) Only the enamel is fractured; no pain or sensitivity to hot and/or cold.
      (2) Treatment is elective (dentist).
   b. Ellis II
      (1) Enamel is fractured and dentin is exposed; hot and/or cold sensitivity often present.
      (2) Management
         (a) Apply a dressing of calcium hydroxide paste over the exposed dentin, and cover it with aluminum foil or dental dry foil.
         (b) All patients should see a dentist for follow-up within 24 hours and be advised to avoid temperature extremes in food and drink.
   c. Ellis III
      (1) Enamel is fractured, and both dentin and pulp are exposed; a pink tinge or drop of blood on the fracture site is characteristic (indicates pulpal exposure). Severe pain is common but may be absent if the neurovascular supply of the tooth has been disrupted.
      (2) This is a true dental emergency, and immediate dental referral is indicated. If a dentist or endodontist is not immediately available, place a piece of moist cotton over the exposed pulp, and cover with a piece of aluminum foil or dental dry foil. Provide tetanus immunization as needed.
2. Alveolar fractures
   a. May be evident (exposed bone) or diagnosed per radiograph (dental panoramic view)
   b. May be associated with dental fractures/avulsions/subluxations
   c. Management
      (1) Immediate dental/oral surgery evaluation for reduction and fixation; wire stabilization is accomplished via the dentition.
      (2) Prophylactic antibiotics (penicillin or clindamycin) and tetanus immunization (if indicated)

3. Avulsed tooth
   a. Permanent tooth
      (1) Holding the tooth by its crown, rinse it gently with saline and immediately replace it in the socket. Do not "brush" the tooth clean, because this will remove the periodontal ligament.
      (2) Viability of the tooth decreases with length of time out of the socket (~1% for every 1 minute out).
      (3) Immediate dental referral for stabilization is indicated.
      (4) Prophylactic antibiotics (penicillin or clindamycin) should be prescribed, and tetanus immunization provided (if indicated).
      (5) If a tooth cannot be immediately reimplanted, the best storage and transplant medium is Hank's solution. Viability of the periodontal ligament can be maintained for 4–6 hours or more in this solution. Milk is a good second choice, as is the inner cheek of the mouth of a reliable patient (caution to avoid aspirating the tooth).
   b. Deciduous/primary tooth (children 6 months to 5 years old) should not be replaced, because alveolar ankylosis may result. These patients should be referred to a pedodontist because space-maintaining procedures and/or appliances are sometimes required.
4. Intra-oral lacerations
   a. Close with absorbable sutures.
   b. Recheck in 24–48 hours.
   c. Although controversial, antibiotic coverage (penicillin or clindamycin) is recommended by many, particularly if the wound is extensive or involves significant amounts of crushed tissue.

B. Hemorrhage may be due to one of the following:

1. Trauma
2. Recent dental manipulation (cleaning, extraction, etc)
3. Gingivitis
4. Blood dyscrasias or coagulopathies

C. Orofacial pain

1. Dental pain
   a. Tooth eruption in adults
      (1) Usually involves the third molars ("wisdom teeth")
      (2) If the gingivae are inflamed (pericoronitis), instruct the patient to irrigate the area with warm saline or hydrogen peroxide.
      (3) If fluctuance and pus are present (pericoronal abscess), make a superficial incision and drain, and place the patient on an antibiotic (penicillin VK or clindamycin).
      (4) Refer these patients to an oral surgeon for definitive treatment (extraction).
   b. Dental caries
      (1) Oral analgesia and dental referral are usually all that is needed.
      (2) Suspect an associated periapical abscess if the patient complains of sharp, severe pain on tooth percussion, especially if the tooth is sensitive to hot or cold. A fluctuant swelling requires incision and drainage, an antibiotic, and warm saline rinses every 2 hours. The patient should be seen by a dentist within 24 hours.

c. Postextraction pain
(1) Periostitis
(a) Pain within 24 hours of extraction
(b) Responds well to oral analgesics
(2) Alveolar osteitis ("dry socket")
(a) Due to loss of clot plus localized osteomyelitis
(b) Clinical presentation: history of a pain-free interval of 2–4 days after extraction followed by sudden onset of excruciating pain unrelieved by analgesics, and a foul breath odor.
(c) Management
i. Anesthetic nerve block
ii. Irrigation of the socket
iii. Packing with iodoform gauze saturated with a medicated dental paste, eugenol, or camphorated phenol provides almost immediate relief.
iv. Dental follow-up in 12–24 hours

d. Acute necrotizing ulcerative gingivostomatitis ("trench mouth")
(1) Only periodontal lesion in which bacteria actually invade nonnecrotic tissue
(2) Caused by *Fusobacterium* and spirochetes
(3) Clinical presentation
(a) The patient complains of pain, a metallic taste, and foul breath, which is frequently accompanied by fever, malaise, and regional lymphadenopathy.
(b) Gingivae are swollen and fiery red; the interdental papillae (tissue between the teeth) are swollen, ulcerated (or "punched out"), and covered with a grayish pseudomembrane.
(4) Management
(a) Warm saline irrigation and hexadine rinse
(b) Antibiotic (penicillin or clindamycin) for patients with fever and lymphadenopathy provides dramatic relief within 24 hours.
(c) Systemic analgesics and topical anesthetic agents can provide the patient sufficient relief to use warm saline rinses, as well as eat and drink.
(d) Dental follow-up is required; a potential complication is the destruction of underlying alveolar bone.

2. Other causes of facial pain
a. Sinusitis (see page 182)
b. Trigeminal neuralgia ("tic douloureux")
c. Herpes zoster (see page 151)
d. Ischemic heart disease
e. TMJ disease (see page 187)
f. Temporal arteritis (see page 550)
g. Cluster headache

D. Systemic diseases with oral manifestations

1. Infections
a. Herpes

b. Coxsackie virus (hand-foot-mouth disease)
c. Gonococcus
d. Syphilis
e. Measles

2. Autoimmune/inflammatory states
   a. Systemic lupus erythematosus: large intraoral ulcerations with necrotic borders
   b. Reiter syndrome (urethritis, arthritis, conjunctivitis, oral ulcers)
   c. Sjögren syndrome
      (1) Autoimmune disease characterized by diminished secretion of the lacrimal and salivary glands
      (2) Symptoms include gritty sensation of the eyes, dry mouth (xerostomia), and diminished sense of taste.
   d. Behcet disease, lesions of inflammatory bowel disease, aphthous ulcers
3. Toxic/metabolic states
   a. Heavy metal poisoning
      (1) Gingival "lead line" in lead poisoning
      (2) Argyria: a blue to bronze discoloration of the oral mucosa in silver poisoning
   b. Gingival hyperplasia
      (1) Diabetes mellitus
      (2) Phenytoin: not related to toxicity; may be secondary to alteration of calcium metabolism
      (3) Nifedipine: not related to toxicity; may be secondary to alteration of calcium metabolism
4. Granulomatous diseases
   a. Tuberculosis: granulomatous ulcerations of the oral cavity
   b. Wegener granulomatosis: gingival hyperplasia with petechiae
5. Benign tumors and tumor-like lesions
   a. Pyogenic granuloma
      (1) A proliferation of capillary-rich connective tissue that develops mostly on the lips and gingiva and commonly occurs secondary to trauma
      (2) Particularly common during pregnancy ("pregnancy tumor")
   b. Epidermoid cyst
6. Blood dyscrasias
   a. Acute leukemia: a marked hyperplastic gingivitis (almost covers the teeth) that has a bluish red discoloration
   b. Thrombocytopenic purpura
      (1) Petechiae of the oral mucosa
      (2) Spontaneous gingival bleeding
7. HIV/AIDS
   a. Oropharyngeal candidiasis
      (1) Painless curd-like plaques on an erythematous base that are easily removed with a tongue blade
      (2) Frequently one of the earliest manifestations of AIDS

b. Hairy leukoplakia
   (1) Asymptomatic white patches with hair-like projections; most commonly located on the lateral aspect of the tongue; cannot be removed with a tongue blade
   (2) Believed to be associated with Epstein-Barr virus
   (3) 80% of patients presenting with these lesions develop AIDS within the next 3 years.
c. Oral Kaposi sarcoma
   (1) Flat bluish red lesions with irregular borders; most commonly found on the hard palate but can be located anywhere in the mouth
   (2) Most common AIDS-related malignancy

HEAD, EAR, EYE, NOSE, AND THROAT DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: An elderly patient with diabetes complains of constant and severe otalgia that is out of proportion to the examination, purulent otorrhea, and redness (inflammatory changes) of both the external auditory canal and periauricular soft tiss

What is the diagnosis?

Scenario B

Presentation: An elderly patient reports nearly a week of prodromal influenza-like illness with dermatomal pain, followed by a unilateral rash of the forehead, upper eyelid, and nose.

What is the diagnosis?

Scenario C

Presentation: A 55-year-old patient with significant myopia (near sightedness) and a history of cataract removal complains of flashing lights with floaters and vision loss. also reports a shadow that varies from light grey to completely black spreads across visual field like a curtain being closed.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: malignant otitis externa

Diagnostic evaluation: Laboratory studies include a CBC (leukocytes may be normal or mildly increased), a serum chemistry panel (to look for glucose intolerance) and erythrocyte sedimentation rate (invariably increased), and cultures of the otorrhea (*Pseudomonas* is causative organism in 95% of cases). MRI and CT are helpful in evaluating the extent of soft-tissue involvement, abscess formation, and intracranial extension. However, they are not reliable for detection of early osteomyelitis.

Management: Management consists of pain control, systemic and ototopic antibiotics that cover *Pseudomonas* (eg, fluoroquinolones), and consult with ENT. Mild to moderate cases may be treated on outpatient basis. More severe cases typically require admission.

Scenario B

Diagnosis: herpes zoster ophthalmicus

Diagnostic evaluation: V1 rash may present at any stage of progression from erythematous macules to vesicles to pustules that crust over within 1 week. Hutchinson sign (herpes zoster lesion at the tip, side, or root of the nose) is prognostic of ocular involvement. Corneal epithelial lesions are best seen with a slit-lamp and fluorescein staining. Herpes zoster ophthalmicus is typically a clinical diagnosis, and laboratory studies are often not necessary. If unclear, a Tzanck smear and Wright stain may be obtained. Nearly 100% of the U.S. population have been infected with varicella-zoster virus by the age of 60.

Management: Management consists of pain control and antiviral treatment (eg, acyclovir, valacyclovir) for 7–10 days. Oral corticosteroids may be considered. Consult with ophthalmology.

Scenario C

Diagnosis: retinal detachment

Diagnostic evaluation: Laboratory studies are often not necessary unless part of a preoperative plan. Plain radiographs, CT, and MRI are also not necessary unless traumatic injury indicates. Ocular ultrasound (of the closed eye) may be diagnostic with experienced operators.

Management: Avoid any pressure on the globe and limit activity. Consult with ophthalmology.

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ABDOMINAL AND GASTROINTESTINAL DISORDERS

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ABDOMINAL AND GASTROINTESTINAL DISORDERS: SELF-ASSESSMENT QUESTIONS

1. The most common cause of oropharyngeal (transfer) dysphagia is:

   (a) Obstructive lesions
   (b) Spasm
   (c) Neuromuscular disorders and inflammatory lesions
   (d) Carcinoma

2. All of the following statements regarding Boerhaave syndrome are accurate except:

   (a) It is usually the result of sudden, violent, repeated vomiting.
   (b) It is a partial-thickness tear of the esophageal wall.
   (c) Associated physical findings with this entity include subcutaneous emphysema and the presence of a mediastinal crunch (Hamman sound).
   (d) Radiographic findings may include a left pneumothorax, a left pleural effusion, mediastinal emphysema, and a widened mediastinum.

3. The most common area for an esophageal foreign body to lodge in an adult is the:

   (a) Aortic arch (T4)
   (b) Cricopharyngeal muscle (C6)
   (c) Lower esophageal sphincter/diaphragmatic hiatus (T10–T11)
   (d) Tracheal bifurcation (T6)

4. Which of the following is the most appropriate therapy for a button battery lodged in the esophagus?

   (a) Observation
   (b) Removal with a Foley catheter
   (c) IV glucagon
   (d) Immediate removal via endoscopy

5. What is the most likely diagnosis in patients with abdominal pain that awakens them at night and is relieved by food intake and ingestion of antacids?

   (a) Duodenal ulcer
   (b) Myocardial ischemia
   (c) Gastric ulcer
   (d) Perforated peptic ulcer

6. Factors that have definitely been demonstrated to predispose to peptic ulcer disease include all of the following except:

   (a) Alcohol ingestion
   (b) Cigarette smoking
   (c) Type O blood
   (d) Use of NSAIDs or aspirin

7. Which of the following statements regarding sucralfate is accurate?

   (a) It neutralizes gastric acid.
   (b) It works most effectively at a gastric pH of 4.5.
   (c) It inhibits secretion of mucus.
   (d) It inhibits pepsin, adsorbs bile acids, and increases mucosal prostaglandin production.

8. The most common cause of small-bowel obstruction is:

   (a) Neoplasms
   (b) Hernias
   (c) Adhesions
   (d) Gallstones

9. All of the following statements regarding volvulus are accurate except:

   (a) Although cecal volvulus can occur at any age, it most commonly affects patients in their twenties and thirties.
   (b) Volvulus is an example of a closed-loop obstruction.
   (c) Sigmoid volvulus occurs in patients with a history of chronic, severe constipation; it primarily affects older, bedridden patients with significant comorbid disease and psychiatric patients of any age.
   (d) Cecal volvulus is more common than sigmoid volvulus.

10. The following statements regarding ulcerative colitis are accurate except:

    (a) Inflammation involves all layers of bowel as well as the mesenteric lymph nodes.
    (b) There is a much higher incidence of colon cancer in patients with ulcerative colitis than in the general population.
    (c) It is characterized clinically by bloody feces associated with crampy abdominal pain.
    (d) Complications include intestinal hemorrhage and toxic megacolon.

11. Unlike small-bowel obstruction due to other causes, an important therapeutic modality in the treatment of small-bowel obstruction due to Crohn disease is:

    (a) Administration of antibiotics
    (b) Nasogastric suction
    (c) Administration of steroids
    (d) IV hydration and correction of electrolyte imbalance

12. All of the following statements regarding pseudomembranous colitis are accurate except:
    (a) It is due to ingestion of broad-spectrum antibiotics (such as the cephalosporins or ampicillin) that alter the gut flora.
    (b) Although they may be delayed, symptoms usually begin within 7–10 days after antibiotic treatment is started.
    (c) Diagnosis is confirmed by identifying the *Clostridium difficile* toxin in the feces.
    (d) Vancomycin is the treatment of choice for patients with mild to moderate disease.

13. A patient presents with the complaint of slight hematochezia and intense rectal pain with bowel movements that linger for several hours and then resolve. The most likely diagnosis is:
    (a) Internal hemorrhoids
    (b) Anal fissure
    (c) Thrombosed external hemorrhoid
    (d) Perirectal abscess

14. A patient presents to the emergency department complaining of frothy, foul-smelling diarrhea and abdominal pain. He says he feels bloated and has a lot of gas. Nine days ago he returned from a hiking trip in Colorado, where he had been drinking water from streams. The most likely cause of his diarrhea is:
    (a) *Giardia lamblia*
    (b) *Vibrio parahaemolyticus*
    (c) *Aeromonas hydrophila*
    (d) *Vibrio cholera*

15. Except for _____, the primary mechanism by which the following bacteria induce illness is by excretion of a toxin.
    (a) *Staphylococcus aureus*
    (b) *Bacillus cereus*
    (c) *Clostridium difficile*
    (d) *Yersinia enterocolitica*

16. An adolescent presents with fever, crampy abdominal pain, and watery diarrhea. Physical examination reveals exquisite tenderness in the right lower quadrant, and a wet mount of the feces reveals WBCs. The most likely organism responsible for this presentation is:
    (a) *Yersinia enterocolitica*
    (b) *Shigella*
    (c) *Clostridium perfringens*
    (d) *Vibrio parahaemolyticus*

17. The organism most often responsible for traveler's diarrhea is:
    (a) *Salmonella*
    (b) Invasive *E coli*
    (c) *Giardia lamblia*
    (d) Enterotoxigenic *E coli*

18. Staining of the feces for leukocytes is helpful in uncovering the probable cause of an acute episode of diarrhea as well as determining appropriate therapy, particularly when this information is combined with a detailed history. Leukocytes are typically present with infections induced by all of the following organisms except:

    (a) *Clostridium difficile*
    (b) Viruses
    (c) Invasive *E coli*
    (d) *Salmonella*

19. All of the following statements regarding scombroid fish poisoning are accurate except:

    (a) It is most commonly associated with ingestion of dark-fleshed or red-muscled fish.
    (b) It results from ingestion of toxins with histamine-like properties that form in improperly preserved or refrigerated fish.
    (c) Epinephrine IM should be administered.
    (d) Symptoms include facial flushing, conjunctival hyperemia, palpitations, nausea, vomiting, and diarrhea.

20. The most common cause of post-transfusion hepatitis is:

    (a) Hepatitis A
    (b) Hepatitis B
    (c) Hepatitis C
    (d) Hepatitis D

21. During the "window period" between the disappearance of HB_sAg and the appearance of anti-HBs, the only marker of hepatitis B infection that may be present in the serum is:

    (a) HB_eAg
    (b) Anti-HB_e
    (c) Anti-HB_c
    (d) HB_cAg

22. The presence of anti-HBs in the serum indicates:

    (a) Ongoing viral replication and high infectivity
    (b) Immunity
    (c) The carrier state
    (d) Low infectivity

23. The presence of marked eosinophilia is typical in patients infected with:

    (a) *Necator americanus*
    (b) *Enterobius vermicularis*
    (c) *Giardia lamblia*
    (d) All of the above

24. A 50-year-old man with a history of chronic alcoholism presents with hematemesis. The bleeding followed an episode of violent and repeated vomiting. It was moderate in quantity and associated with pain on swallowing but has resolved on its own. The diagnostic study of choice for evaluating this patient is:

    (a) A chest radiograph
    (b) A barium esophagogram
    (c) A CT of the chest
    (d) Endoscopy

25. An 8-year-old child presents with a foreign body sensation in his throat and relates that he "accidentally swallowed" a quarter a couple of hours before presentation. The PA view of the chest reveals the flat surface of the coin. Where is the coin located?

    (a) In the esophagus
    (b) In the trachea
    (c) Unable to tell from the information given

26. All of the following types of viral hepatitis may be associated with development of a chronic infectious state except:

    (a) Hepatitis B
    (b) Hepatitis C
    (c) Hepatitis delta
    (d) Hepatitis E

27. All of the following statements regarding *Helicobacter pylori* are accurate except:

    (a) It is a gram-negative, spiral-shaped organism that colonizes only gastric mucosa.
    (b) It has been associated with the development of peptic ulcer disease (particularly duodenal ulcers).
    (c) In patients with peptic ulcer disease who are infected with this organism, eradication of the infection effectively eliminates the ulcer diathesis.
    (d) Single-agent therapy with either bismuth or amoxicillin is very effective in eliminating this organism.

28. A 38-year-old woman presents with epigastric pain of several hours duration. She states that it started shortly after eating a meal of fried shrimp and onion rings. She recalls that she has had similar pain in the past, but states that it has never lasted this long. She is moderately obese and has a history of hypercholesterolemia for which she is taking gemfibrozil. Her past medical history is otherwise noncontributory, and there is no prior history of abdominal surgery. Examination reveals epigastric and right upper quadrant tenderness. She is afebrile, and her rectal examination is negative. The most useful initial test for evaluating this patient is:

    (a) A CT scan of the abdomen
    (b) Biliary scintiscanning (HIDA, DISIDA)
    (c) Abdominal ultrasonography
    (d) Plain radiographs of the abdomen

29. The most accurate study for confirming diagnosis of acute cholecystitis is:

(a) A CT scan of the abdomen
(b) Biliary scintigraphy (HIDA, DISIDA)
(c) Abdominal ultrasonography
(d) Plain radiographs of the abdomen

30. All of the following statements regarding anal canal tumors are accurate except:

(a) They are defined as tumors that occur proximal to the dentate line.
(b) Presenting symptoms may include rectal bleeding, decrease in fecal caliber, constipation, and weight loss.
(c) They represent 80% of all anorectal tumors.
(d) They are slow to metastasize and have a low-grade malignant potential.

31. Most acute episodes of diarrhea are caused by:

(a) Viruses
(b) Enterotoxin-producing bacteria
(c) Invasive bacteria
(d) Parasites

32. Which of the following statements regarding diarrhea in patients with AIDS is false?

(a) Cytomegalovirus and *Cryptosporidium* are the two most common causes of diarrhea in these patients.
(b) Multiple organisms are responsible in up to 25% of cases.
(c) It is not a self-limited disease in these patients.
(d) Empiric antibiotics should be administered immediately.

33. Which of the following statements regarding chest pain due to esophageal spasm is least accurate?

(a) It is frequently triggered by emotional upset.
(b) It can be precipitated by drinking extremely hot or cold liquids.
(c) It can produce a dull discomfort, localized pressure, or a sensation of severe squeezing pressure across the middle of the chest.
(d) Pain associated with esophageal spasm can be distinguished from that which occurs in association with myocardial ischemia by evaluating its response to nitroglycerin; pain of esophageal spasm is not relieved by nitroglycerin.

34. The finding of _____ on plain abdominal radiographs is strongly suggestive of mesenteric infarction.

(a) Ileus
(b) Pneumatosis intestinalis
(c) Gasless abdomen
(d) Sentinel loop

35. Evaluation of an elderly patient who presents with severe abdominal pain reveals a diagnosis of nonocclusive mesenteric ischemia. In the absence of peritonitis/necrotic bowel, definitive therapy for this patient consists of:

(a) Systemic heparinization
(b) Urokinase infusion
(c) Intra-arterial papaverine infusion
(d) Exploratory laparotomy

36. In a patient with acute pancreatitis, which of the following laboratory reports is most likely to be associated with the highest mortality rate according to the Ranson criteria?

(a) Amylase 800 units/L, AST 300 units/L, WBC count 3,000/mm^3
(b) Lipase 1100 units/L, AST 350 units/L, WBC count 17,000/mm^3
(c) LDH 300 units/L, ALT 200 units/L, glucose 50 mg/dL
(d) LDH 400 units/L, glucose 400 mg/dL, WBC count 18,000/mm^3

37. Hepatitis D is caused by:

(a) An RNA virus
(b) A defective RNA virus
(c) A DNA virus
(d) A defective DNA virus

38. Which of the following laboratory findings is not consistent with a diagnosis of alcoholic hepatitis?

(a) AST > ALT
(b) AST and ALT levels in the thousands
(c) Prolonged prothrombin time
(d) Increased bilirubin and alkaline phosphatase levels

39. The toxic metabolite responsible for producing hepatic necrosis in patients who overdose on acetaminophen is:

(a) APAP-mercapturate and cysteine
(b) APAP-sulfate
(c) APAP-glucuronide
(d) N-acetyl-p-benzoquinone imine (NAPQI)

ANSWERS

1. c	8. c	15. d	22. b	29. b	36. d
2. b	9. d	16. a	23. a	30. d	37. b
3. c	10. a	17. d	24. d	31. a	38. b
4. d	11. c	18. b	25. a	32. d	39. d
5. a	12. d	19. c	26. d	33. d	
6. a	13. b	20. c	27. d	34. b	
7. d	14. a	21. c	28. c	35. c	

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. UPPER GASTROINTESTINAL BLEEDING

A. Evaluation

1. Evaluation of the patient with a presumed upper GI bleed must start with an assessment of hemodynamic stability.
   a. Unstable patients require immediate resuscitation and stabilization.
      (1) Two large-bore (at least 18-gauge) IV lines for crystalloid infusion (2 L bolus); continue fluids until vital signs have stabilized or 40 mL/kg have been given (adults).
      (2) Cardiac monitoring.
      (3) Oxygen administration as needed.
      (4) Blood should be sent immediately for hemoglobin and hematocrit, platelet count, prothrombin time/INR, and type and crossmatch.
      (5) Immediate transfusion of type-specific or crossmatched blood (4 units minimum) if available (type O if type-specific blood unavailable).
      (6) May need reversal of coagulation abnormalities; the following regimen is a Class 1C recommendation:
         (a) Hold anticoagulation therapy
         (b) Vitamin K 10 mg (may be given IV slowly)
         (c) Fresh frozen plasma or
         (d) Prothrombin complex concentrate or
         (e) Activated recombinant factor VII
   b. Patients who remain unstable require immediate consult with a gastroenterologist for immediate endoscopy.
2. Evaluation continues with an assessment of bleeding.
   a. Determination of the location of bleeding facilitates appropriate therapy.
   b. The following are predictive of an upper GI source of bleeding:
      (1) Melena (likelihood ratio [LR] +25)
      (2) Nasogastric lavage with presence of blood or "coffee grounds" (LR +9.6)
         (a) Not necessary if history of hematemesis
         (b) Nasogastric lavage remains controversial; its use is not universally accepted.
         (c) A "negative lavage" (no blood returned) does not have an adequate negative likelihood ratio to exclude upper GI bleeding, although a lavage returning nonbloody, bilious fluid makes it unlikely.
      (3) Serum BUN:creatinine ratio >30 (LR +7.5)
      (4) Hematocrit <20 (LR +2.6)
      (5) Use of anticoagulants (LR +2.3)
   c. The following factors decrease the likelihood of an upper GI source of bleeding:
      (1) Blood clots in feces (LR 0.05)
      (2) History of lower GI bleed

B. Blatchford Score

1. Accurately predicts the need for urgent or emergent intervention in a patient with acute upper UG bleeding (may still require validation in U.S. hospitals)

2. Does not require nasogastric lavage
3. Based on hemoglobin, BUN, systolic blood pressure, pulse, melena, syncope, and history of hepatic disease or cardiac failure
4. Specific scores
   a. Patients with upper GI bleed and a score of 0 have an LR of 0.02 of needing urgent intervention.
   b. Those with a score of ≤2 have an LR of 0.08 of needing urgent intervention.

C. Specific treatment of ruptured varices

1. Immediate therapy: octreotide or vasopressin infusion
   a. Octreotide is considered more effective than vasopressin and has fewer adverse effects.
   b. Vasopressin has no proven mortality benefit but may be of use in the exsanguinating patient.
2. Placement of a balloon tamponade device may be used as a temporary measure while awaiting endoscopy.
3. Endoscopic band ligation or sclerotherapy
4. If endoscopic intervention is unsuccessful, perform angiography with embolization of the gastric vein.
5. Transjugular intrahepatic portosystemic shunt placement for refractory bleeding helps prevent rebleeding.
6. Surgical therapy is rarely used but may be only option in continuously bleeding patient.
7. Antibiotic therapy: ceftriaxone or ciprofloxacin IV reduces risk of rebleeding and infectious complications.

D. Treatment of bleeding due to peptic ulceration

1. Peptic ulceration is the most common cause of upper GI bleeding, even in patients with history of varices.
2. Duodenal ulcers (most common): 29% of upper GI bleeds
3. Gastric ulcers: 16% of upper GI bleeds
4. Combined, gastric and duodenal ulcers cause 45%–50% of all upper GI bleeding.
5. Management
   a. Volume replacement with normal saline or lactated Ringer's via two large-bore IV lines
   b. Oxygen at 6–8 L/min
   c. Foley catheter placement to monitor volume resuscitation
   d. Lavage
      (1) Can be used to monitor blood loss and prepare patients for endoscopy but is not helpful in stopping the bleeding or preventing its recurrence.
      (2) To prevent complications, only room-temperature solutions (normal saline or water) should be used; pneumoperitoneum must first be excluded.
      (3) Many gastroenterologists do not consider performance of gastric lavage necessary to establish the diagnosis or monitor bleeding.
   e. Erythromycin 250 mg IV push over 20 minutes provides satisfactory conditions for endoscopy without the need for nasogastric lavage.
   f. IV proton-pump inhibitor therapy should be given to any patient with active upper GI bleeding, because the benefits in acute ulcer bleeding have been well established.

(1) IV formulations of pantoprazole and esomeprazole are available.

(2) No proven effect on mortality but may decrease re-bleed rate

6. Consult with a gastroenterologist and a general surgeon is prudent in any patient with significant upper GI bleeding (see above).
7. If the patient does not respond to the above measures and bleeding persists, early or emergent endoscopic evaluation may be needed for both localization of the bleeding site and treatment. Although arteriography is also useful in locating the bleeding site (if the rate of bleeding is ≥0.5 mL/min), it has largely been supplanted by endoscopy, which is usually more accurate; it is currently reserved for situations in which endoscopy is unavailable or nondiagnostic.
8. Surgical intervention is indicated for patients who do not respond to medical therapy and endoscopic hemostasis.
9. Older patients are more likely to bleed, re-bleed, require surgery to control bleeding, and require blood products.

II. ESOPHAGEAL DISORDERS

A. Functional anatomy

1. The esophagus originates in the hypopharynx at the level of the cricoid cartilage (C6) and terminates in the cardia of the stomach (T11). It is ~25 cm (10″) long and is divided into three segments (helpful in locating foreign bodies and abnormalities of contiguous structures on radiograph).
   a. Upper (cervical) = 4–5 cm
   b. Middle (thoracic) = 15–20 cm
   c. Lower (abdominal) = 2–3 cm
2. Layers of the esophagus
   a. Inner mucosa: Esophagitis involves both the inner mucosa and submucosa; if severe and prolonged, esophagitis may result in scarring and stricture formation. Treatment is dilatation. If the cause is eliminated, recurrent stricture is not a problem.
   b. Submucosa: Because there is no serosa, perforation of the submucosa extends into surrounding mediastinal structures and leads to a diffuse, malignant, often rapidly progressive and fatal mediastinitis.
   c. Muscle layers: If the two muscle layers are split by bougienage or by repeated dilatation, they scar, which leads to stricture formation. Treatment is dilatation, but strictures recur.
3. Cricopharyngeal muscle (upper esophageal sphincter) is located at the level of C6.
4. Muscle composition of the esophagus
   a. Upper third is striated muscle.
   b. Middle third is smooth and striated muscle.
   c. Distal third (and remaining GI tract) is smooth muscle.
5. Neuromuscular anatomy
   a. Extrinsic nervous system
      (1) Spinal accessory nerve innervates the cervical esophagus.

(2) Vagus nerve innervates the remainder of the esophagus. Stimulation of this parasympathetic nerve during esophageal intubation and endoscopy causes bradycardia.

(3) Sympathetic fibers from the cervical and thoracic ganglia also innervate the esophagus. Stimulation causes dysphagia and referred pain to the chest and epigastrium.

b. Intrinsic nerve supply (Auerbach and Meissner plexuses, which are contained between the muscle layers of the esophageal wall) is altered in motor disorders (achalasia, diffuse spasm) and destroyed by corrosives and some collagen vascular disorders (scleroderma, those associated with Raynaud syndrome).

B. Dysphagia

1. Dysphagia is defined as "difficulty swallowing"; it signifies the presence of organic pathology of the esophagus in nearly all cases. Odynophagia means "pain on swallowing"; it often indicates the presence of an inflammatory or infectious lesion.
2. Oropharyngeal (transfer) dysphagia (difficulty swallowing)
   a. Symptoms occur within the first 2 seconds of swallowing (cough, choking, drooling, repeated swallowing attempts)
   b. Neuromuscular disorders account for most cases. (Difficulty swallowing liquids [particularly cold liquids] suggests a neuromuscular disorder.)
      (1) Neuromuscular causes (80% of cases)
         (a) Stroke (most common cause)
         (b) Polymyositis and dermatomyositis
         (c) Scleroderma (>50% complain of dysphagia)
         (d) Myasthenia gravis (reversible with edrophonium)
         (e) Multiple sclerosis, amyotrophic lateral sclerosis, Parkinson disease
         (f) Lead poisoning
      (2) Inflammatory/infectious causes
         (a) Pharyngitis (*Streptococcus*, *Candida*, herpes)
         (b) Oropharyngeal abscess
      (3) Infectious/neuromuscular causes
         (a) Poliomyelitis
         (b) Diphtheria
         (c) Botulism
         (d) Rabies
         (e) Tetanus
   c. Cancer of the tongue, pharynx, or larynx may also cause oropharyngeal dysphagia.
3. Upper esophageal dysphagia
   a. Difficulty swallowing occurs within the first 2–4 seconds and is caused by obstructive lesions in most cases.
      (1) Intrinsic causes of luminal narrowing are esophageal webs (which are associated with iron deficiency anemia, Plummer-Vinson syndrome), and carcinoma.
      (2) Extrinsic compression can be produced by numerous conditions, eg, thyroid enlargement, Zenker diverticulum, left atrial enlargement, aortic aneurysm.

b. Dysphagia due to obstructive lesions is typically progressive (starts with solid foods and eventually progresses to liquids).

4. Lower esophageal dysphagia (difficulty swallowing)
   a. Symptoms occur 4–10 seconds after the bolus is swallowed and are usually worse with solids.
   b. Clinical presentation: patients complain of a substernal "sticking" sensation and are usually able to accurately pinpoint the location.
   c. Lower esophageal dysphagia is usually due to luminal narrowing. This narrowing can be either constant (eg, strictures, carcinoma) or intermittent (eg, spasm).
      (1) Carcinoma is the most common cause of lower esophageal dysphagia. Difficulty swallowing solids occurs initially and progresses to difficulty swallowing semisolids and finally liquids.
      (2) Achalasia is characterized by marked increase in the resting pressure of the lower esophageal sphincter and absent peristalsis in the body of the esophagus. Dysphagia is the most common presenting complaint.
         (a) Patients are usually 20–40 years old.
         (b) A diagnostic clue is that dysphagia occurs with both solids and liquids.
         (c) The patient may regurgitate undigested material; no acidic taste.
      (3) Esophageal strictures result from esophageal reflux. There is a history of heartburn before the onset of dysphagia, which is worse with solid foods and is constant; reflux-induced spasm without stricture produces intermittent dysphagia.
      (4) "Steakhouse syndrome" is characterized by intense discomfort that develops shortly after swallowing a large piece of meat. It can occur in patients with a normal esophagus but is more commonly associated with one of the following if the problem has occurred repeatedly:
         (a) Carcinoma
         (b) Stricture
         (c) Schatzki ring (a fibrous, diaphragm-like stricture near the gastroesophageal junction); is actually an esophageal web
   d. Lower esophageal dysphagia and chest pain: gastroesophageal reflux disease or ischemic heart disease? Chest pain arising from the esophagus may mimic chest pain due to myocardial ischemia because of similar segmental innervation of the heart and esophagus; both may present with ST segment abnormalities on ECG. The history is by far the most useful information in differentiating these two entities.
      (1) A substernal burning sensation (heartburn/pyrosis) is a common complaint in patients with esophageal reflux. However, a dull discomfort, localized pressure, or severe squeezing pain across the middle of the chest may be seen when reflux is associated with esophageal spasm.
      (2) Exacerbation of symptoms with stooping, lying, or leaning forward suggests reflux. Postural exacerbation of pain is uncommon with ischemic heart disease.
      (3) Water brash (a hypersalivation response) is commonly associated with reflux.
      (4) Radiation of pain into the abdomen occurs more often in patients with reflux than in those with coronary artery disease. Radiation into both arms is rarely seen in reflux.
      (5) A feeling of fullness after meals and relief of chest pain with antacids are key points in the history. This history is rarely present in patients with ischemic heart disease.

(6) Diffuse esophageal spasm without reflux may appear clinically similar to myocardial ischemia. Differentiating features include the following:
(a) Emotional upset frequently triggers spasm.
(b) Chest pain from esophageal spasm occurs spontaneously and most often at rest.
(c) Diffuse spasm may be precipitated by swallowing liquids of extreme temperatures (hot or cold).
(d) Esophageal spasm and myocardial ischemia are both relieved by nitroglycerin. Although the latter generally responds more quickly, response to nitroglycerin is not useful in distinguishing these clinical entities.
(e) If a barium swallow is done, "corkscrew" appearance.

5. Esophagitis (inflammation of the esophagus)
a. Clinical presentation
(1) Chest pain, dysphagia, odynophagia, or those of reflux
(2) Chest pain is typically constant, acute in onset, and unresponsive to treatment with antacids.
b. Etiology
(1) Most common cause is gastroesophageal reflux disease (GERD)
(2) Infections: typically present with severe odynophagia; may have dysphagia to solids and liquids
(a) Fungal (*Candida albicans*)
(b) Viral (herpes simplex, varicella zoster, cytomegalovirus)
(c) Bacterial (mycobacterium)
(d) Immunosuppressed patients are more likely to have herpes simplex virus or cytomegalovirus as cause because of prophylactic treatment of fungal infections.
(3) Radiation
(4) Corrosive agents (alkalis, acids)
(5) Pill esophagitis: antibiotics (especially doxycycline and tetracycline), bisphosphonates, antiinflammatory agents, potassium chloride, and iron sulfate
c. Management
(1) Fluconazole is the drug of choice for esophageal candidiasis.
(2) Immunocompromised patients should be assumed to have herpes simplex and should receive acyclovir.

C. Esophageal trauma

1. Etiology
a. Swallowing (foreign body, caustic agent)
b. Instrumental (surgery, bougienage, rigid endoscopy, intubation, etc); *iatrogenic perforation is the most common cause of esophageal perforation.*
c. Chest trauma (open, closed)
d. Sudden, violent, and usually repeated increase in the intra abdominal pressure against a weakened esophageal wall; most common cause is violent and repeated vomiting/retching.
(1) Mallory-Weiss syndrome

(a) A partial thickness tear (mucosal lesion) of the distal esophagus or gastric cardia associated with dysphagia, odynophagia, and upper GI bleeding.

(b) Clinical presentation: the prominent symptom is bleeding, which is usually mild to moderate and usually resolves spontaneously.

(c) Predisposing factors

i. Alcoholism

ii. Hiatal hernia

iii. Gastritis/esophagitis

(d) Diagnostic study of choice is endoscopy.

(2) Boerhaave syndrome

(a) Specifically refers to spontaneous esophageal rupture, usually due to retching

(b) A full-thickness tear (perforation) typically of the left posterolateral aspect of the distal esophagus (an intrinsically weak area)

(c) Clinical presentation

i. The prominent symptom is severe, lancinating chest pain.

ii. Physical findings include subcutaneous emphysema, mediastinal "crunch" (Hamman sound), a left pneumothorax and/or pleural effusion, and possibly epigastric tenderness.

(d) Diagnostic evaluation

i. Chest radiograph: characteristic findings include a left pneumothorax, a left pleural effusion, mediastinal emphysema, and a widened mediastinum.

ii. An esophagram using water-soluble oral contrast confirms the diagnosis.

- Water-soluble contrast causes less inflammatory response (mediastinitis) than barium but has a lower sensitivity. If an initial water-soluble contrast esophagram is negative, it should be followed by a barium study if suspicion persists.
- Option following water-contrast or barium swallow if nondiagnostic is CT scan in the noncritical patient whose diagnosis is unclear.

(e) Management

i. Fluid resuscitation

ii. Broad-spectrum IV antibiotics

iii. Boerhaave syndrome is a surgical emergency; do not delay surgical consult for CT or other advanced imaging.

(f) This lesion produces the most malignant type of mediastinitis (acid burn and bacterial infection). Morbidity and mortality, due to shock and septicemia, occur very rapidly (<48 hours from the time of perforation). Hence, immediate repair is indicated.

2. Diagnosis of esophageal trauma is confirmed by water-soluble contrast swallow or endoscopy.
3. Contraindications for insertion of a nasogastric tube
   a. Posterior laryngeal lacerations
   b. Esophageal tears
   c. Foreign bodies (associated with esophageal wall trauma)
   d. Caustic ingestions
   e. Near-total obstruction of the esophagus secondary to stricture
   f. Basilar skull or midface fractures

D. Esophageal foreign body ingestion

1. Most esophageal impactions (80%) occur in children and involve true foreign bodies (coins, buttons, marbles, etc); obstruction occurs less frequently in adults and is usually the result of a food impaction. Foreign bodies tend to lodge or impact at the sites where esophageal narrowing occurs.
   a. Cricopharyngeus muscle (C6): most common site in children <4 years old
   b. Adjacent to the aortic arch (T4)
   c. Lower esophageal sphincter/diaphragmatic hiatus (T10–T11): most common site in adults
2. Clinical presentation
   a. Patients <16 years old
      (1) Vomiting
      (2) Gagging
      (3) Choking (also exclude tracheal foreign body, especially if associated with stridor, wheezing, coughing, or other signs of respiratory distress)
      (4) Neck or throat pain
      (5) Dysphagia
      (6) Foreign body sensation in the chest
      (7) Refusal to eat
   b. Patients >16 years old
      (1) Anxiety and discomfort
      (2) Foreign body sensation
      (3) Substernal chest pain (sometimes)
      (4) Progressive dysphagia and inability to handle secretions
      (5) Odynophagia
   c. Usually, the patient can accurately locate the position of the impacted esophageal foreign body.
3. Diagnostic evaluation
   a. Order radiographs of the neck (AP and lateral) and chest films (PA and lateral) in symptomatic patients.
      (1) A flat object (such as a coin) will be oriented in the frontal (coronal) plane if it is located in the esophagus, and the PA or AP view will reveal the flat surface of the coin; if the coin is in the trachea, it will be oriented in the sagittal plane (reflecting the angle of the coin required to pass through the vocal cords), and the PA or AP view will reveal the edge of the coin.
      (2) Radiopaque esophageal foreign bodies will show on chest radiographs.
      (3) Some fish bones are radiolucent and not visible on plain radiographs. This is also true of plastic objects and toothpicks.
      (4) Radiographic evaluation of asymptomatic patients with suspected or known ingestions should also be considered, because many patients are asymptomatic; up to one-third of children with esophageal foreign bodies are asymptomatic. Newer recommendations include use of CT scan to demonstrate foreign bodies not visualized on plain radiographs.
      (5) If the esophageal foreign body is not detected by neck and chest radiographs, an esophagram or endoscopy should be performed.

(a) Endoscopy: the diagnostic modality of choice (if readily available), because it can be used to both visualize and remove the foreign body.

(b) Esophagram: if a perforation is suspected, a watersoluble contrast agent should be selected. If aspiration is a concern, barium should be used. If both perforation and aspiration are considerations, a nonionic contrast media should be used. Before the procedure, a nasogastric tube should be inserted above the obstruction to prevent aspiration of unswallowed liquids that collect there.

(c) Barium should not be used if subsequent endoscopy is anticipated because it may limit or obscure visualization of the esophageal foreign body by the endoscopist.

4. Although objects do occasionally lodge or become impacted in the esophagus (particularly those with sharp edges), most will pass as a matter of course. Low-risk objects (blunt, small, nonmagnetic, or button battery) can be watched in the esophagus for up to 24 hours. Once an object has passed the gastroesophageal junction, the probability of eventual passage is >90%. The foreign body usually arrives at the rectum in 3–5 days. As long as the patient is asymptomatic, management in these cases is expectant.
5. Management
   a. Because most esophageal foreign bodies are in the cervical esophagus, removal is usually accomplished with a laryngoscope and Magill forceps or with an endoscope.
   b. Food impactions
      (1) A distal esophageal food impaction can occasionally be dislodged with IV glucagon (frequently causes nausea and vomiting), sublingual nitroglycerin, or nifedipine. IV glucagon relaxes smooth muscle and decreases lower esophageal sphincter pressure. After a test dose to exclude hypersensitivity, administer 1 mg IV; an additional 2 mg may be given if there is no relief in 20 minutes. Nitroglycerin and nifedipine are rarely used because of the high incidence of adverse effects and limited efficacy.
      (2) Gas-forming agents (eg, effervescent granule packets, carbonated beverages) may also be used to dislodge an esophageal food impaction. They work by producing CO_2, which increases intraluminal pressure and pushes the food bolus into the stomach. These agents should be avoided in patients with chest pain (possible perforation) and in those with symptoms >24 hours duration.
   c. The practice of removing an impacted, smooth foreign body (such as a coin) with a Foley or Fogarty catheter requires a cooperative patient, fluoroscopic guidance, and an object that has been present for <24–72 hours. This procedure is best avoided; the foreign body may slip from the esophagus to the trachea and lead to respiratory compromise.
   d. Bougienage has been used safely to push the object into the stomach.
   e. Alkaline disk batteries (button batteries)
      (1) 90% pass through the GI tract in 48–72 hours without any untoward effect.
      (2) If the patient is asymptomatic and the battery has already passed into the stomach, expectant management (observation at home) is adequate. If the patient develops symptoms, eg, abdominal pain, emergency surgical removal may be required.
      (3) If a button battery becomes lodged, it usually does so in the esophagus.
      (4) A button battery lodged in the esophagus is associated with severe morbidity and must be removed immediately even if the patient is asymptomatic. The corrosive action of these batteries can produce esophageal burns, necrosis, and perforation within 4–6 hours of ingestion.

f. Single magnets can generally be managed expectantly with serial radiographs. Multiple magnets should be removed promptly, because they can attract across layers of bowel and lead to perforation, obstruction, or volvulus.

g. Impacted foreign bodies that cannot be dislodged or removed by direct visualization must be extracted surgically (~1% of all foreign body ingestions).

h. Sharp or pointed objects, as well as objects >5 cm long and >2 cm wide, must be removed endoscopically. They should be removed before they pass the pylorus, because 15%–35% will cause perforation, usually in the region of the ileocecal valve.

i. After an esophageal foreign body has been removed, esophageal function should be thoroughly evaluated (including motility studies) to ensure that there is no underlying pathology that led to the obstruction. This is particularly true of adults with meat impactions; underlying pathology of the esophagus is present up to 97% of the time.

III. PEPTIC ULCER DISEASE

A. Pathophysiology

1. Peptic ulcers are mucosal defects that extend beyond the muscularis mucosae. They are most commonly located along the lesser curvature of the stomach or in the first portion of the duodenum.
   a. Gastric ulcers are thought to be due to damage to the gastric mucosal barrier itself, which allows diffusion of hydrogen ions from the lumen to the gastric mucosa and results in ulceration.
   b. Duodenal ulcers are usually associated with hypersecretion of gastric acid (by an enlarged parietal cell mass) and with markedly increased gastric emptying that overwhelms the capacity of the duodenum to neutralize gastric acid, and results in damage to the duodenal mucosal barrier. *Helicobacter pylori* causes ~90% of all duodenal ulcers.
2. Uncommon sites of peptic ulceration
   a. Distal esophagus
   b. Ectopic gastric mucosa with a Meckel diverticulum
   c. Margins of surgical anastomoses ("marginal ulcers")
3. A stress ulcer is not the same as a peptic ulcer, ie, the mucosal lesion of a stress ulcer does not extend through the muscularis mucosae. Stress ulcers are most commonly found in the body and fundus of the stomach and are a common cause of gastric bleeding (hemorrhagic gastritis).

B. Predisposing factors

1. Peptic ulcer disease is considered to have two main causes: *H pylori* infection and NSAID usage
2. Zollinger-Ellison syndrome or other gastrin-secreting tumors cause 1%
3. Cigarette smoking
4. Bile salts
5. Emotional stress
6. Prolonged use of corticosteroids
7. Caffeinated beverages (coffee, soda, tea)

C. Clinical presentation

1. The pain associated with gastric and duodenal (peptic) ulcers is visceral in nature and, therefore, is vague (not well defined). It is generally felt in the midline and is typically described as gnawing, aching, or burning.
2. Gastric pain is generally perceived at the midline or to the left of the epigastrium.
3. Duodenal bulb pain is usually perceived to the right of the midepigastrium. With posterior penetration, the pain may radiate straight through to the back.
4. The patient presents with epigastric pain that may be easily confused with the substernal chest pain of myocardial ischemia. A significant diagnostic clue is the character of the pain: a patient with peptic ulcer disease will describe the pain as a burning sensation; the MI patient generally will not.

D. Diagnostic evaluation

1. Definitive diagnosis is generally not made in the emergency department.
2. Peptic ulcers may be diagnosed with an upper GI or endoscopy. Endoscopic evaluation with biopsy is key to differentiating malignant from benign gastric ulcers and should be suspected in patients >50 years old as well as in those with markers of malignancy.
3. Infection with *H pylori* can be diagnosed noninvasively via serology or the urea breath test, or by tests that require endoscopy (rapid urea/C10 test, histology).

E. Management of uncomplicated peptic ulcer disease is generally on an outpatient basis and may be started in the emergency department.

1. Advise patients to avoid substances that exacerbate gastric and duodenal ulcers (smoking, alcohol, NSAIDs, etc). A bland diet with frequent feedings has not been demonstrated to be effective and is probably unnecessary.
2. Provide pain relief and facilitate ulcer healing with one of the following agents:
   a. Antacids
      (1) Provide pain relief and accelerate healing by neutralizing gastric acid
      (2) Dose: 30 mL at bedtime and at 1 and 3 hours after meals
      (3) Their major drawback is how frequently they must be taken, which decreases compliance. They may also cause constipation (aluminum salts) or diarrhea (magnesium salts). In addition, antacids decrease the absorption of certain drugs (warfarin, digoxin, several anticonvulsants, and some antibiotics).
   b. Histamine (H_2)-antagonists
      (1) Promote healing by inhibiting gastric acid secretion
      (2) The major advantage of these drugs is their convenient dosing (once or twice daily).
   c. Proton-pump inhibitors
      (1) Promote healing by blocking the secretion of gastric acid
      (2) Inhibit the H^+/K^+ ATPase enzyme system (the "proton pump") of parietal cells, thereby preventing the release of hydrogen ions into the gastric lumen
      (3) Indicated for the short-term treatment of duodenal and gastric ulcers, and usually reserved for those in whom H_2-blockers have not been effective
      (4) Agents
         (a) Omeprazole
         (b) Lansoprazole

(c) Pantoprazole (available in IV form; has been shown to ↓ bleeding from peptic ulcers)

(d) Esomeprazole and rabeprazole are newer agents.

d. Misoprostol

(1) A synthetic prostaglandin E_1 analogue that acts in a way similar to that of the naturally occurring prostaglandins secreted by the gastric mucosa in response to injury.

(2) Indicated only for the prevention of NSAID-induced gastric ulcers in high-risk patients (the elderly, those with concomitant debilitating disease or a history of ulcers)

(3) Can cause spontaneous abortion and is, therefore, contraindicated in pregnant women and in women of childbearing age who are not using reliable contraceptive measures

e. Sucralfate: drug of choice for erosive esophagitis secondary to large "stuck" pills (eg, doxycycline, docusate)

(1) Works locally at the ulcer site, where it binds to the base of the ulcer, thereby protecting it from the adverse effect of gastric acid; also adsorbs bile acids, inhibits pepsin activity, and increases mucosal prostaglandin production

(2) Dosage is 1 g orally qid on an empty stomach. It works best in an acid environmen (pH <3.5); thus, simultaneous administration of antacids should be avoided.

f. Bismuth compounds (bismuth subsalicylate/subcitrate): diminish pepsin activity, increase mucus secretion, create a barrier to additional acid damage of ulcerated surfaces, augment prostaglandin synthesis, slow hydrogen ion diffusion through the mucosal barrier, and have a bactericidal effect on *H pylori*; used in quadruple therapy

g. Eradication of *H pylori* (if present)

(1) *H pylori* is a gram-negative spiral-shaped organism that colonizes only gastric-type epithelium and is associated with the development of peptic ulcer disease; it is responsible for ~95% of duodenal ulcers and ≥70% of gastric ulcers.

(2) In patients with peptic ulcer disease who are infected with *H pylori*, eradication of the infection effectively eliminates the ulcer diathesis. Relapses and recurrences ar markedly reduced, and the need for chronic suppressive therapy with H_2-blockers is eliminated.

(3) Management of patients with suspected or confirmed peptic ulcer disease should, therefore, include a referral for diagnostic testing to determine if infection with *H pylori* is present.

(4) Patients with confirmed infections should be started on a regimen that is effective i eradicating *H pylori*. Triple therapy consists of a proton-pump inhibitor, amoxicilli and clarithromycin. Metronidazole can be substituted for amoxicillin in penicillin-allergic patients.

F. Complications

1. The most common complications are bleeding (20% of ulcer patients), perforation (7%), and gastric outlet obstruction.
2. Elderly patients are at greatest risk of hemorrhage.
3. Ulceration into an artery can lead to life-threatening hemorrhage.
4. Perforation

a. Exposure of the peritoneal cavity to gastric or duodenal contents produces a chemical peritonitis that, in the absence of prompt and adequate treatment, rapidly progresses to bacterial peritonitis.

b. Clinical presentation

(1) History of ulcer-like pain in weeks before presentation helpful in making diagnosis.

(2) Sudden onset of abdominal pain with guarding and rebound is characteristic of anterior perforations; back pain is characteristic of posterior perforation of a duodenal ulcer.

(3) Pain may radiate to shoulder and or chest.

(4) Posterior, perforated duodenal ulcer may appear similar to pancreatitis (but the serum lipase will be normal or slightly increased initially if the perforation is adjacent to the pancreas), or the perforation may actually cause pancreatitis.

c. Diagnostic evaluation

(1) Absence of free air on radiographs does not exclude the diagnosis.

(a) Anterior perforations: only 60%–70% demonstrate free air.

(b) Posterior perforations: no free air will be evident because the posterior duodenum is located retroperitoneally.

(2) Diagnosis may require CT scan of abdomen or endoscopy.

d. Management

(1) IV fluids (normal saline or lactated Ringer's) and electrolyte replacement

(2) Nasogastric tube drainage

(3) Broad-spectrum IV antibiotics

(4) Immediate surgical consult

5. Gastric outlet obstruction

a. When an ulcer heals, it can form a scar that blocks the pyloric outlet. The resulting obstruction can result in gastric dilation, vomiting, dehydration, and a hypokalemic, hypochloremic metabolic alkalosis.

b. Clinical presentation

(1) Upper abdominal pain and vomiting (most common)

(2) Early satiety

(3) Recent weight loss

(4) A succussion splash (a splashing sound elicited by gently rocking the abdomen)

c. Diagnostic evaluation: an upright abdominal radiograph typically reveals a prominently dilated stomach shadow with a large air–fluid level.

d. Management

(1) Constant nasogastric suctioning

(2) Fluid replacement and correction of electrolyte abnormalities

(3) Admission for further evaluation and definitive management

(4) Anticholinergic agents are contraindicated, because they may aggravate gastric distention by decreasing gastric motility.

IV. PERFORATED VISCUS

A. Gallbladder perforation (rare)

1. Gallstone obstruction of the cystic or common bile duct → gallbladder distention → vascular compromise → gangrene → perforation
   a. More commonly, gallstones erode through the gallbladder wall, cystic duct, or common duct and produce fistulas between the gallbladder and another portion of the GI tract, rather than perforate directly into the peritoneal cavity.
   b. Gallstone ileus occurs when a large gallstone enters the small intestine through such a fistula. It usually lodges in the terminal ileum, producing a small-bowel obstruction. May see pneumobilia on abdominal radiograph.
   c. Gangrene of the gallbladder (with subsequent perforation) can occur in the absence of stone formation (acalculous cholecystitis), especially in diabetic patients.
2. Patients at increased risk
   a. Older patients
   b. Those with diabetes
   c. Those with atherosclerotic cardiovascular disease
   d. Those with a history of gallstones and repeated cholecystitis
   e. Those with a hemolytic disorder (sickle cell disease)
3. Classic clinical scenario: An older man with a fever appears ill and has a tender right upper quadrant mass on physical examination. A careful history reveals one or more risk factors. In addition, a nonalcoholic may give a past history of jaundice or pancreatitis, which should suggest common duct stones. Laboratory studies reveal leukocytosis, possibly an increased bilirubin, and slightly increased serum amylase. On abdominal radiograph, a stone may be seen free in the abdomen. Subhepatic or subphrenic abscesses can form after a gallbladder perforation and cause restricted movement of the right leaf of the diaphragm.

B. Small-bowel perforation

1. Etiology of jejunal rupture
   a. Drugs (enteric-coated potassium tablets)
   b. Infection (eg, typhoid, tuberculosis)
   c. Tumor
   d. Strangulated hernia
   e. Regional enteritis (Crohn disease)
2. A jejunal perforation causes a chemical peritonitis that is more severe than that caused by an ileal perforation because the pH of the jejunal contents is higher (pH 8), and there is also a greater number of enzymes present in this part of the bowel. Ileal perforations are associated with considerable bacterial contamination; these small-bowel perforations can rapidly wall off (particularly in patients with Crohn disease), so that early evaluation may reveal only localized findings.
3. Mortality varies directly with the extent of peritoneal soiling and time delay to diagnosis and treatment.

C. Large-bowel perforation

1. Etiology
   a. Carcinoma
   b. Diverticulitis
   c. Colitis
   d. Foreign body
   e. Diagnostic instrumentation
2. Clinical presentation
   a. The signs and symptoms of large-bowel perforations are largely due to sepsis and, therefore, are usually slower in onset than those produced by small-bowel perforations.
   b. The patient appears septic and has a feculent breath odor. The abdomen is distended, and the patient has been unable to pass feces. When a nasogastric tube is inserted and hooked to suction, fecal material is seen in the aspirate. Radiograph often demonstrates free air and small-bowel obstruction.

D. Management of a known or suspected perforated viscus

1. IV fluid replacement with normal saline or lactated Ringer's; large volumes (up to 12 L over 24 hours) may be required because of third spacing.
2. Bladder catheterization (to monitor urine output/volume status)
3. Nothing by mouth; nasogastric tube drainage
4. Broad-spectrum IV antibiotics
5. Immediate surgical consult (delay in surgical intervention significantly increases mortality)

I. ACUTE ABDOMEN

A. Appendicitis

1. **The most common indication for emergency surgery; appendicitis is also the most common surgical emergency seen in pregnancy, and the most common cause of emergency abdominal surgery in children.**
2. **Although all age groups are affected, the highest incidence is in patients 10–30 years old (particularly males).**
3. **Pathophysiology**
   a. **Obstruction of the appendiceal lumen (the primary inciting event) → increased intraluminal pressure and distention → vascular compromise of the appendiceal wall and bacterial invasion**
   b. **Etiologies of appendiceal obstruction include:**
      (1) **Fecalith (most common)**
      (2) **Enlarged lymphoid follicles**
      (3) **Inspissated barium**
      (4) **Worms**
      (5) **Granulomatous disease**
      (6) **Tumors**
      (7) **Adhesions**
      (8) **Dietary matter (seeds)**

4. Clinical presentation (listed in order of decreasing frequency)
   a. History
      (1) Abdominal pain (with migration to right lower quadrant increases likelihood)
      (2) Anorexia
      (3) Nausea and vomiting
      (4) Fever and chills (minimal statistical association between temperature >99°F [37.2°C] and appendicitis)
      (5) Diarrhea (can be a particularly prominent symptom in very young children and patients with pelvic appendices; often results in misdiagnosis as acute gastroenteritis)
   b. Physical evaluation
      (1) Abdominal tenderness
      (2) Percussion or rebound tenderness
      (3) Rectal tenderness (examination not useful to diagnose or exclude)
      (4) Cervical motion tenderness (28%–34%)
      (5) Psoas sign (right lower quadrant pain on passive extension of right hip)
      (6) Obturator sign (right lower quadrant pain on passive internal rotation of the flexed right hip)
      (7) Rovsing sign (right lower quadrant pain on palpation of the left lower quadrant)
      (8) Right lower quadrant pain on rectal examination (retrocecal appendicitis)
   c. Visceral pain is the first symptom to develop; it is dull and vague in character. In nonpregnant patients, pain usually begins in the periumbilical area and then, over time, moves to the right lower quadrant. Where the pain eventually migrates is determined by the actual location of the appendix; radiation of pain to the flank is suspicious of a retrocecal appendicitis. (Remember that kidney stone pain usually comes on suddenly; retrocecal appendiceal pain usually has a gradual onset). In pregnant patients, the appendix moves laterally and superiorly as the uterus enlarge so be suspicious of flank or right upper quadrant pain. If the appendix is located nea the uterus, ovary, or tubes, there will be pain on cervical motion. (Patients with pelv inflammatory disease are generally seen later in the course of their illness, have few GI symptoms, and have a more increased sedimentation rate). Anorexia, nausea, an vomiting usually begin after the onset of pain. A sudden decrease in pain followed b a dramatic increase suggests perforation.
   d. Unfortunately, one-third to one-half of patients do not present "classically" (migratory abdominal pain and the typical associated signs and symptoms); the pain can be anywhere.
   e. Be particularly suspicious in very young and very old patients; their symptoms are frequently vague, and clinical presentation is less classic. Delayed diagnosis with resulting perforation and increased morbidity and mortality is common in these age groups.
5. Diagnostic evaluation
   a. May provide additional information even when the history and physical examinatio are classic; they can also be helpful in less typical cases when the diagnosis remains doubt.
   b. Routine studies

(1) Pregnancy test: should be done in all women of childbearing age to exclude ectopic pregnancy

(2) CBC: a WBC count >10,000/mm^3 is statistically associated with appendicitis but has such poor sensitivity and specificity that it is of almost no clinical utility.

(3) Urinalysis: pyuria and hematuria may be seen if the inflamed appendix lies in close proximity to the ureter; a few WBCs and RBCs in the urine are characteristic.

(4) Plain abdominal radiographs (an option to be considered only if CT is unavailable): a calcified fecalith in the right lower quadrant is very suggestive of appendicitis but is present in only 2%–22% of cases.

c. Additional studies may be indicated when the diagnosis remains uncertain and should be chosen in consultation with the evaluating surgeon. Availability and usefulness of these studies may be institution-dependent.

(1) Abdominal CT is generally considered the radiographic procedure of choice for diagnosing acute appendicitis in men and nonpregnant women. Data suggest that abdominal CT is >97% sensitive and 98%–100% specific for acute appendicitis. (In addition, abdominal CT may unmask other cases of abdominal pain.) Abdominal CT is often performed with IV contrast and oral or rectal contrast; presence of periappendiceal fat stranding is the most specific radiographic finding for appendicitis. Unenhanced (noncontrast) CT is also highly sensitive and specific.

(2) Diagnostic laparoscopy has high sensitivity and specificity. It is clearly indicated in equivocal cases of surgical abdominal pain, and its use as a diagnostic tool is increasing because laparoscopic appendectomies are now performed more frequently than open surgical appendectomies.

(3) Graded compression ultrasonography has a sensitivity of ~40% and a specificity of ~90%, and it may be especially valuable for evaluating children and pregnant patients. It is noninvasive, without radiation exposure, safe in pregnancy, rapid, inexpensive, and rarely requires sedation in children. Visualization of a noncompressible, immobile appendix >6 mm in diameter is very suggestive of the diagnosis. Furthermore, an alternative definitive diagnosis can be established in up to 50% of patients when appendicitis is absent. The disadvantages of ultrasound are that it is not always available, it is operator-dependent, and it has a very poor sensitivity in the presence of perforation.

(4) Lifetime risk of all cancers from one CT abdomen is calculated at 0.14% in neonates and 0.06% in adults.

(5) In children, use ultrasound to confirm suspected acute appendicitis but not definitely exclude the diagnosis. Recommendation to follow ultrasound with CT of abdomen and pelvis if diagnosis is still highly suspected. (*ACEP Clinical Policy:* Critical Issues in the Evaluation and Management of Patients with Suspected Appendicitis)

(6) MRI appears to be a useful modality in pregnant women (to avoid ionizing radiation) in whom ultrasound is inconclusive.

6. Differential diagnosis

a. Mesenteric adenitis

b. *Yersinia* gastroenteritis

c. Pelvic inflammatory disease

d. Ectopic pregnancy

e. Ovarian cyst

f. Pyelonephritis
g. Crohn disease
h. Diverticulitis

7. Management
   a. Nothing by mouth
   b. Establish an IV line
   c. Obtain early surgical consult. (Delay in laparoscopy or surgical intervention → gangrene → perforation → increased morbidity and mortality, particularly in the very young and very old.)
   d. If a decision to operate is reached, administer prophylactic parenteral antibiotics; they decrease the incidence of postoperative wound infection and, in patients who have perforated, they decrease the incidence of postoperative abscess formation. Acceptable regimens:
      (1) Piperacillin/tazobactam
      (2) Cefoxitin
      (3) Cefotetan
      (4) Ampicillin, gentamicin, and metronidazole/clindamycin
   e. Judicious amounts of parenteral narcotic may be administered for pain relief and to facilitate diagnostic evaluation. (*ACEP Clinical Policy:* Critical Issues for the Initial Evaluation and Management of Patients Presenting with a Chief Complaint of Nontraumatic Acute Abdominal Pain)
   f. Recent literature confirms that delaying appendectomy in stable patients is a safe alternative to immediate surgery.
   g. Several studies have compared operative versus nonoperative treatment of acute appendicitis with conflicting results. A 2011 Cochrane Review considers the data inconclusive.

B. Bowel obstruction

1. Etiology
   a. Small-bowel obstruction
      (1) Adhesions (most common)
      (2) Hernia (second most common)
      (3) Neoplasms (lymphoma, adenocarcinoma)
      (4) Intussusception (common in children <2 years old)
      (5) Gallstones (gallstone ileus)
      (6) Bezoars
      (7) Crohn disease
      (8) Radiation enteritis
      (9) Foreign bodies
   b. Large-bowel obstruction (much more common in the elderly)
      (1) Neoplasm
         (a) Most common cause of large-bowel obstruction
         (b) Remember that tumors on the left side cause obstruction, while tumors on the right side present with bleeding.
      (2) Diverticulitis (one of the most common causes of large-bowel obstruction in adults)

(3) Volvulus (sigmoid and cecal)
(4) Fecal impaction
(a) The elderly
(b) Narcotic and laxative abusers
(c) The mentally challenged
(d) Nursing-home patients

2. Pathophysiology
a. Mechanical obstruction refers to partial or complete compromise of the bowel lumen.
b. Closed-loop obstruction refers to a segment of bowel that is blocked proximally and distally. It results in rapid increase of intraluminal pressure. Intestinal ischemia, infarction, and perforation can ultimately develop. Examples include:
(1) Volvulus (twisting of a bowel loop on its mesenteric axis)
(2) Herniation of a bowel loop in the omentum or mesentery or a loop of intestine caught in a hernia sac
(3) A complete colonic obstruction (such as carcinoma or impaction) in the presence of a competent ileocecal valve causes progressive distention of the involved bowel and creates a form of closed-loop obstruction; if the ileocecal valve is incompetent, decompression can occur by reflux into the ileum.
c. Peristalsis is initially increased early in the course of bowel obstructions (hyperactive bowel sounds). Passage of feces and flatus often continues in the first few hours after an obstruction occurs. With persistent complete obstruction, the bowel loses its ability to contract rigorously, bowel sounds become infrequent, and the passage of feces/flatus stops.
d. Strangulation occurs most often in the small bowel and is due to vascular compromise that leads to infarction. In the large bowel, strangulation can occur with a volvulus, because it is a closed loop obstruction and vascular supply is quickly compromised.

3. Clinical presentation
a. Pain
(1) Crampy, intermittent, and poorly localized pain is typical.
(2) A change in the quality of the pain (greater intensity, more constant) may indicate that a complication has developed.
b. Vomiting
(1) The more proximal the obstruction, the sooner it begins.
(2) If it is bilious, the obstruction is distal to the pylorus.
(3) Feculent emesis is associated with distal ileal and large-bowel obstruction and, if long-standing, may be due to infarcted bowel.
c. Abdominal distention: the more distal the obstruction, the more pronounced the distention.
d. Tympany may be present on percussion.
e. Abdominal tenderness
(1) Mild, diffuse tenderness is typical.
(2) A tender mass may be palpated with a closed-loop obstruction.
(3) Severe localized tenderness or rebound tenderness suggests the possibility of gangrenous or perforated bowel.

4. Diagnostic evaluation
   a. Radiographs of the abdomen should include supine and upright films and are the first choice for imaging.
   b. Small-bowel obstruction
      (1) Abdominal radiographs are diagnostic in 30%–70% (specificity ~50%).
      (2) Small-bowel diameter >3 cm
      (3) Air fluid levels
      (4) Stepladder appearance of dilated loops
      (5) "String of pearls" appearance is a late finding.
      (6) Cause of obstruction rarely identified on plain radiographs.
   c. CT of the abdomen and pelvis
      (1) The American College of Radiology considers CT an appropriate first choice for imaging; emergency medicine sources usually suggest abdominal radiographs.
      (2) Oral contrast not usually required, because fluid in bowel acts as contrast.
      (3) IV contrast helps diagnose strangulation.
      (4) Transition zone often identified.
      (5) Sensitivity 94%; specificity for small-bowel obstruction is 95% for high-grade obstructions but only 50% for low-grade obstructions.
   d. Gas patterns in small- and large-bowel obstructions
      (1) Colon gas is distinguished from gas in the small bowel by its peripheral location and the presence of haustrations that do not involve the entire transverse diameter of the bowel.
      (2) Small-bowel gas is more central in location, and one can see valvulae conniventes that involve the entire transverse diameter of the small bowel. They are spaced closer together than the haustrations of the large bowel.
   e. Large-bowel obstruction
      (1) Abdominal radiographs unreliable in diagnosis (may be only 84% sensitive and 72% specific)
      (2) Dilation of colon >5 cm with >10 cm at cecum
      (3) CT of abdomen and pelvis is considered modality of choice.
5. Management
   a. Small-bowel obstruction
      (1) IV fluid resuscitation
      (2) Correction of electrolyte abnormalities
      (3) Nasogastric tube decompression is mainstay of conservative therapy; no benefit for long intestinal tubes.
      (4) Broad-spectrum antibiotic coverage may be of benefit.
      (5) Early surgical consult: patients with pneumoperitoneum, peritonitis, and sepsis require immediate surgical intervention.
   b. Large-bowel obstruction
      (1) Volume resuscitation and correction of electrolyte abnormalities.
      (2) Patients with perforated bowel, sepsis, etc, require immediate surgery.
      (3) Colonic dilatation >12 cm or of prolonged duration may require immediate intervention.
      (4) Nasogastric decompression if vomiting present.

C. Volvulus

1. Volvulus is a closed-loop obstruction that results from the twisting of a mobile segment of bowel on its mesocolon; it is responsible for approximately 10%–13% of colonic obstructions in the United States.
2. Sigmoid volvulus is more common (60%) than cecal volvulus (40%).
3. Epidemiology and pathophysiology
   a. Sigmoid volvulus occurs almost entirely in two patient populations: elderly, bedridden patients with debilitating comorbid diseases and patients of any age with profound neurologic or psychiatric illness. These patients invariably have a history of chronic severe constipation that leads to an elongated (redundant) sigmoid colon.
   b. Cecal volvulus results from incomplete embryologic fixation of the cecum, ascending colon, and terminal ileum to the posterior abdominal wall. Although it is seen in patients of all ages, it is most common in patients in their twenties and thirties.
4. Clinical presentation of sigmoid and cecal volvulus
   a. Sudden onset of crampy lower abdominal pain
   b. Nausea, vomiting, obstipation
   c. Diffuse abdominal tenderness
   d. Progressive abdominal distention
   e. Tympany
   f. Gangrenous bowel in 20% of patients with cecal volvulus
5. Diagnostic evaluation
   a. Radiographic findings: a markedly dilated single loop of colon
   b. Sigmoid volvulus: bent "inner tube" appearance with dilated bowel segment doubled back; the proximal large bowel is dilated.
   c. Cecal volvulus: the loop is usually seen in the mid or upper abdomen toward the left but may be anywhere; single dilated segment of cecum with a kidney or "coffee bean" shape. The distal large bowel is collapsed, and the small bowel is dilated.
6. Management
   a. Initial measures are the same as those for bowel obstruction (IV hydration, nasogastric tube decompression, and broad-spectrum antibiotics).
   b. Sigmoid volvulus
      (1) Surgical consult and nonoperative reduction using a rectal tube via the sigmoidoscope or barium enema. Because recurrence is common (90%), this is usually followed by an elective resection if the patient's underlying medical condition can tolerate it.
      (2) If nonoperative management fails or strangulation is suspected, immediate operative reduction is required.
   c. Cecal volvulus: early surgical reduction, because this is a closed-loop obstruction.

D. Mesenteric vascular ischemia/infarction

1. Most commonly affects patients who are >50 years old and have a history of cardiovascular disease
2. Mortality rate is 50% overall (but rises to ≥70% once infarction has occurred) and reflects the difficulty in making an early diagnosis as well as the presence of significant underlying disease.
3. Etiology

a. Most common cause is arterial embolus (50% of cases). Another 25% are caused by arterial thrombosis.

b. Nonocclusive mesenteric ischemia accounts for 20% of cases. Common pathway is mesenteric vasoconstriction due to low flow states with decreased cardiac output. Associated conditions are CHF, cardiogenic shock, septic shock, etc, and medications that cause splanchnic vasconstriction such as digoxin, vasopressors. It is common in hospitalized patients.

c. Mesenteric venous thrombosis is responsible for 5%–15% of cases and most commonly occurs in association with a hypercoagulable state (eg, polycythemia vera, antithrombin III deficiency). Affected patients are usually younger in age, and up to 60% of them have a history of peripheral deep-venous thrombosis.

4. Predisposing factors for

a. Arterial occlusion

(1) Dysrhythmias (particularly atrial fibrillation)

(2) Atherosclerotic heart disease

(3) Valvular heart disease

(4) Recent MI

b. Venous thrombosis

(1) History of prior thromboembolic events

(2) Hypercoagulable states

c. Nonocclusive ischemia

(1) Use of diuretics or vasoconstrictive medications or digitalis

(2) Hypotension

(3) CHF

(4) Dialysis

5. Clinical presentation

a. The patient is typically middle-aged or elderly and presents with severe abdominal pain, fever, and tachycardia; sudden onset suggests arterial occlusion, while insidious onset suggests venous thrombosis or nonocclusive ischemia.

b. A history of similar, spontaneously resolving pain episodes after meals ("intestinal ischemia") is not uncommon.

c. Early on, the pain is poorly localized and out of proportion to findings on physical examination. Diarrhea is common, and the feces frequently test positive for guaiac. Grossly bloody feces may also be seen. Other commonly associated symptoms are anorexia, nausea, and vomiting. Abdominal distention and peritoneal signs are late findings and signal the presence of bowel infarction.

6. Diagnostic evaluation

a. Helical CT angiography is the primary diagnostic modality for patients with a high clinical index of suspicion. Catheter angiography is used for equivocal cases.

b. CBC reveals hemoconcentration (increased hematocrit) and leukocytosis (WBC count is often >15,000/mm^3).

c. Serum amylase is usually moderately increased (lipase is normal).

d. Serum phosphate may be increased.

e. Serum lactate is nearly 100% sensitive when bowel infarction is present but lacks specificity and is not often increased without infarction.

f. Arterial blood gases often reveal a metabolic acidosis, particularly late in the disease course. Presence of metabolic acidosis that cannot otherwise be explained should prompt investigation for mesenteric ischemia in appropriate patients.

g. Plain abdominal and upright chest radiographs are usually the first radiographic studies to be obtained, although they are often normal (especially early on). Findings may include:

(1) Ileus

(2) Small-bowel obstruction

(3) Gasless abdomen

(4) Irregular thickening of the bowel wall ("thumbprinting"—although rarely seen, strongly suggests intestinal infarction)

(5) Gas in the bowel wall (pneumatosis intestinalis) or portal venous system (although rarely seen, strongly suggests intestinal infarction)

h. Abdominal ultrasound and MRI are not useful in the initial evaluation.

7. Management

a. Initial stabilization measures

(1) IV of normal saline or lactated Ringer's to correct volume deficit

(2) Supplemental oxygen

(3) Nasogastric tube to decompress stomach and bowel

(4) Initial laboratory studies, type and cross, cultures, and radiographs

(5) Broad-spectrum IV antibiotics

(6) Correction of precipitating or predisposing causes of ischemia (eg, CHF, dysrhythmias)

(7) Immediate surgical consult

(8) If pressors are required, positive inotropes are preferred. Avoid α-agonists.

(9) If angiography is performed, papaverine may be infused to dilate mesenteric arteries.

b. Specific treatment measures depend on the underlying cause of the ischemia and if peritoneal signs or necrotic bowel are present.

(1) Nonocclusive mesenteric ischemia: unless peritonitis or necrotic bowel is present, treatment is nonoperative; papaverine alone is considered definitive therapy in these patients.

(2) Mesenteric venous thrombosis: immediate anticoagulation with heparin is the initial treatment of choice and, in the absence of peritonitis and necrotic bowel, may be the only treatment given.

(3) Acute mesenteric arterial occlusion (embolic and thrombotic): initial treatment may be papaverine, while plans are made for revascularization. Papaverine should be continued postoperatively to treat vasospasm of the smaller branches of the superior mesenteric artery.

(4) Immediate surgery is indicated for:

(a) Patients with peritonitis

(b) Resection of necrotic bowel

E. Hernias

1. Definitions
   a. Hernia: the protrusion of a structure from its normal position into another through an opening that is either congenital or acquired
   b. External hernia: protrudes to the outside (eg, umbilical)
   c. Internal hernia: protrudes within the body (eg, diaphragmatic)
   d. Incisional hernia: protrudes through a previous incision
   e. Reducible hernia: protruding contents can be pushed back in
   f. Irreducible (incarcerated) hernia
      (1) Protruding contents cannot be moved back into place
      (2) Seen most often when large contents herniate through a small defect
   g. Strangulated hernia: vascular compromise of herniated contents
   h. "Sports hernia": refers to groin pain related to participation in athletics but is not an anatomic hernia
2. Epidemiology
   a. Men are 8 times more likely than women to develop a hernia, and 20 times more likely to require surgical repair.
   b. Lifetime risk of developing a groin hernia is 25% in men.
   c. Women present at an older age than men.
   d. 96% of groin hernias are inguinal; 4% are femoral.
3. Types
   a. Inguinal hernia
      (1) Direct
         (a) Protrudes directly through the floor of Hesselbach triangle
         (b) Results from relaxation/weakening of the abdominal musculature
         (c) Increased frequency with age
         (d) Rarely incarcerates
      (2) Indirect
         (a) Protrudes through the internal inguinal ring lateral to the inferior epigastric vessels
         (b) Represents a congenital defect (incomplete closure of processus vaginalis)
         (c) Most common hernia occurring in both sexes
         (d) Most common in younger patients
         (e) Frequently incarcerate, especially in infancy
   b. Femoral hernia
      (1) Protrudes below the inguinal ligament and the femoral vessels in the femoral canal
      (2) More common in women
      (3) Frequently incarcerates (40% present as acute incarceration)
   c. Umbilical hernia
      (1) Usually presents as a lump
      (2) Common in newborns
      (3) Most close spontaneously by the age of 2–3 years

d. Obturator hernia
   (1) Protrudes through the obturator foramen into the medial thigh
   (2) Most occur in older women
   (3) Patients present with pain and decreased sensation along the medial aspect of the thigh to the knee.

4. Diagnostic evaluation
   a. Inguinal: ultrasonography is considered the best initial modality in patients with high clinical suspicion but negative findings on examination.
   b. Femoral: ultrasonography may help differentiate inguinal from femoral hernias, but CT scan may be required.

F. Ileitis and colitis

1. Crohn disease (terminal ileitis, granulomatous ileocolitis, regional enteritis)
   a. Pathology
      (1) A chronic inflammatory disease that involves all layers of the bowel as well as the mesenteric lymph nodes
      (2) Any segment of the GI tract from the mouth to the anus may be affected.
      (3) The disease is discontinuous with normal areas of bowel ("skip areas") between one or more involved areas; the ileum is involved in most cases.
      (4) The thickened bowel wall leads to narrowing of the lumen, which frequently results in obstruction.
      (5) Longitudinal deep ulcerations are characteristic; with progression of the disease, a "cobblestone" appearance of the mucosa results from crisscrossing of these ulcers with intervening normal mucosa.
      (6) Noncaseating granulomas are present in ≥50% of specimens and are helpful (but not necessary) in making the diagnosis.
      (7) Perianal complications occur in 90% of cases and may be the initial presenting symptom in many patients.
   b. Epidemiology
      (1) Onset of disease common in teens and early twenties but can occur at any age
      (2) Four to eight times more common in the Jewish population
      (3) Genetic predisposition
      (4) Etiology remains undetermined.
   c. Clinical presentation
      (1) Abdominal pain and diarrhea (inflammation of ileum and/or colon)
         (a) Past history of recurrent abdominal pain, diarrhea, and fever for several years
         (b) Nocturnal diarrhea may be a diagnostic clue.
         (c) Weight loss is common.
      (2) Right lower quadrant pain and tenderness (acute inflammation of the terminal ileum only)
         (a) Looks like appendicitis ("pseudoappendicitis"): anorexia, diarrhea, vomiting, fever, leukocytosis
         (b) Diagnostic clue: occult blood and fecal leukocytes favor regional enteritis

(3) Extraintestinal manifestations are seen in one-quarter to one-third of cases and may be the presenting and/or dominant manifestation of the disease process.
  (a) Arthritic (peripheral arthritis, ankylosing spondylitis)
  (b) Vascular (vasculitis, arteritis, thromboembolic disease)
  (c) Hepatobiliary (gallstones, pericholangitis, chronic active hepatitis)
  (d) Dermatologic (erythema nodosum, pyoderma gangrenosum)
  (e) Ophthalmic (uveitis, episcleritis, iritis, conjunctivitis)
  (f) Renal calculi occur with increased frequency due to increased intestinal absorption of oxalic acid.

d. Complications
  (1) Perianal (abscess, fissure, fistula, rectovaginal fistula, rectal prolapse)
  (2) Intestinal (obstruction, abscess, fistula, stricture, perforation, hemorrhage)
  (3) Toxic megacolon is an infrequent complication of Crohn disease and is associated with massive GI bleeding in >50% of cases.
  (4) Malignancies of both the small and large bowel are three times more common in patients with Crohn disease than in the general population.

e. Diagnostic evaluation
  (1) Previously undiagnosed patients require radiologic, colonoscopic, and histologic confirmation to establish the local and systemic extent of the disease,
  (2) Stable patients with mild disease may be evaluated as an outpatient with close follow-up.

f. Management
  (1) Make a diligent effort to exclude associated small-bowel obstruction, because early institution of nasogastric suction, IV fluids, and steroids may reduce inflammatory edema, restore bowel patency, and avoid the need for emergency surgery.
  (2) Fluid and electrolyte replacement (especially those patients with a history of anorexia, vomiting, and diarrhea)
  (3) Indications for admission
    (a) Dehydration or metabolic/electrolyte disturbances
    (b) Severe exacerbation of the primary illness
    (c) Acute complications (eg, obstruction, hemorrhage, peritonitis)
  (4) Outpatient therapy for mild cases
    (a) Bed rest, analgesics, antidiarrheal agent
    (b) Milk products should be avoided; they frequently aggravate diarrhea in these patients, in whom the incidence of lactose intolerance is high.
    (c) Steroids: beginning therapy with (or changing the dosage of) should be done in conjunction with the patient's primary physician, because these agents have many adverse effects and are ineffective as maintenance therapy.
    (d) Mesalamine, a 5'-ASA derivative is preferred over sulfasalazine, because it has fewer adverse effects.
    (e) Antibiotics
      i. May be of use in Crohn disease
      ii. Ciprofloxacin or metronidazole show some benefit.

(f) Azathioprine and its metabolite 6-mercaptopurine (immunosuppressive agents) are useful:

i. As steroid-sparing agents (allow patients to decrease their dose of prednisone)

ii. In patients with refractory disease and contraindications to surgery

iii. In patients with enterocutaneous or enteroenteric fistulas

(g) Anti-TNF therapies (infliximab, adalimumab, certolizumab pegol) may be helpful in severe cases.

2. Ulcerative colitis

a. Definition and pathology

(1) Ulcerative colitis is a chronic inflammatory and ulcerative disease of the colon and rectum, most often characterized clinically by bloody diarrhea and crampy abdominal pain.

(2) Inflammation is generally limited to the mucosa and submucosa; the muscular layer and serosa are usually spared.

(3) Unlike the "skip lesions" of Crohn disease, mucosal involvement in ulcerative colitis is continuous and uniform; it always begins in the rectum and can remain limited to this area or spread proximally to involve the upper segments of the colon.

(4) Chronic inflammation leads to the formation of crypt abscesses, epithelial necrosis, and mucosal ulceration.

b. Epidemiology closely approximates that of Crohn disease.

c. Clinical presentation

(1) Occurs intermittently with complete remission between attacks

(2) Mild disease in 60% of cases

(a) <4 bowel movements/day

(b) No systemic symptoms

(c) Few extraintestinal manifestations

(3) Severe disease (fulminant colitis) in 15% of cases

(a) >6 bowel movements/day

(b) Fever, tachycardia, weight loss, anemia

(c) Extraintestinal manifestations (essentially the same as those seen with Crohn disease)

d. Complications

(1) Hemorrhage

(a) Most common

(b) However, massive hemorrhage is infrequent (occurs in only 2%–3% of patients).

(2) Toxic megacolon (occurs more commonly with ulcerative colitis than with Crohn disease)

(a) Clinical picture: The patient appears severely ill and has abnormal vital signs (hypotension, tachycardia, and fever). The abdomen is distended, tender, and tympanitic. Laboratory studies reveal leukocytosis with anemia, electrolyte imbalance, and hypoalbuminemia. Plain radiograph of the abdomen reveals dilatation of the colon with a colonic diameter >6 cm.

(b) Management

i. Nasogastric suction, IV fluids, and broad-spectrum antibiotics

ii. IV steroids (hydrocortisone or methylprednisolone)

iii. Frequent abdominal examinations and radiographs with surgery (colectomy) if no improvement in 24–48 hours

(3) Perforation (occurs more often during first episodes of colitis)

(4) Obstruction (due to stricture)

(5) Perianal fistulas and abscesses (occur less commonly with ulcerative colitis than with Crohn disease)

(6) Carcinoma of the colon (incidence is 15 times greater in patients with ulcerative colitis than in the general population and correlates with the duration and extent of disease)

e. Diagnostic evaluation

(1) The most sensitive method for establishing the diagnosis of ulcerative colitis and determining the extent of disease is a sigmoidoscopy.

(2) Diagnosis of toxic megacolon is usually obvious on plain radiographs.

f. Management

(1) Mild to moderate disease

(a) 5′-ASA derivative such as mesalamine or olsalazine

i. Mainstay of therapy

ii. Therapeutic properties are because of its 5-aminosalicylic acid (5′-ASA) component.

(b) Corticosteroids may also be needed, either as supplemental therapy or as alternative therapy when mesalamine is ineffective.

(c) 5′-ASA and corticosteroid enemas are useful for patients with ulcerative proctitis and left-sided colitis.

(d) Supportive therapy (supplemental iron and a lactose-free diet); antidiarrheal agents should be avoided because they are usually ineffective and can precipitate toxic megacolon.

(e) Azathioprine, cyclosporine, and 6-mercaptopurine are used in patients who do not respond to steroid therapy.

(2) Severe ulcerative colitis (inpatient therapy)

(a) IV fluids and correction of electrolyte abnormalities

(b) Nasogastric tube

(c) IV steroids, cyclosporine, or infliximab

(d) Broad-spectrum antibiotics active against coliforms and anaerobes (ampicillin, clindamycin, or metronidazole)

(e) Surgical consult if toxic megacolon suspected

3. Pseudomembranous enterocolitis

a. Definition: an inflammatory bowel disorder characterized by the formation of yellowish exudative pseudomembranous-like plaques that overlie and replace necrotic intestinal mucosa, particularly in the area of the rectosigmoid

b. Etiology and epidemiology

(1) Ingestion of broad-spectrum antibiotics alters the gut flora → proliferation of *Clostridium difficile*, a cytopathic toxin-producing bacterium (emerging strain NAP-1/027 has high association with fluoroquinolone usage)

(2) Any antibiotic can cause pseudomembranous colitis, but fluoroquinolones, clindamycin, cephalosporins, and penicillins are the most common.

(3) Nosocomial transmission among hospitalized patients via hands of healthcare workers (contaminated with *C difficile* toxin) who are caring for these patients also occurs, even in the absence of antimicrobial therapy.

(4) Traditionally considered a disease of frail, elderly, hospitalized patients, it is now recognized in those who have not been exposed to hospital environment or antibiotics. The CDC warns of transmission by close contact with infected persons in nontraditional patients (such as the young and otherwise healthy individuals); suggests person-to-person spread.

c. Clinical presentation

(1) The patient presents with crampy abdominal pain, fever, and watery diarrhea (which may be bloody).

(2) Symptoms generally begin 7–10 days after antibiotics are started but may occasionally be delayed several weeks after antibiotics have been discontinued.

d. Diagnostic evaluation

(1) The presence of *C difficile* toxin in the feces confirms the diagnosis.

(2) If this assay is not readily available, colonoscopy or sigmoidoscopy can be used to establish a tentative diagnosis.

e. Management

(1) Discontinue antibiotics and institute supportive therapy with IV fluids and electrolyte replacement.

(2) Oral metronidazole considered first choice for mild to moderate disease.

(3) Oral vancomycin is first choice for severe infection and refractory infections.

G. Irritable bowel syndrome (IBS)

1. Definition: a state of disturbed intestinal motility (without anatomic cause) that is functional in nature and diagnosed only after more serious causes are excluded
2. Epidemiology
   a. Patients are usually 20–40 years old.
   b. Women are affected more often than men (ratio is 2:1).
   c. A familial predisposition is present.
   d. Psychiatric disease may coexist. Depression and anxiety disorder are common.
   e. It is exacerbated by stress.
   f. Patients with IBS have demonstrable disturbance of rhythmic electrical activity in the intestine.
3. Classic clinical scenario
   a. The patient is usually a young woman who presents with recurrent episodes of altered bowel function (diarrhea or constipation) with or without associated abdominal pain.
   b. The abdominal pain is described as crampy or achy in character and is confined to the lower abdomen. It generally occurs in association with constipation and is relieved by defecation or the passage of gas.
   c. Extracolonic symptoms (eg, bloating, belching, gastroesophageal reflux) are common.

d. Presence of fever, abdominal mass, weight loss, and rectal bleeding are not associated with IBS and suggest a more serious diagnosis.

e. Examination findings are most often nonspecific but may include vague lower abdominal tenderness and a palpable feces-filled sigmoid colon.

f. Laboratory evaluation is unremarkable.

4. Diagnostic evaluation

a. IBS is a diagnosis of exclusion.

b. Diagnosis is difficult to make in the emergency department. Patients are often discharged with diagnosis of "abdominal pain, etiology uncertain." If the diagnosis is suspected, patients should be referred to primary care provider for additional evaluation.

c. The Rome Criteria were developed to standardize research protocols: diagnosis of IBS requires recurrent abdominal pain or discomfort for at least 3 days per month in the last 3 months associated with at least two of the following:

(1) Improvement with defecation

(2) Onset associated with change in frequency of defecation

(3) Onset associated with change in form/appearance of feces

5. Management

a. Directed toward relief of symptoms

(1) Constipation predominant: may benefit from high-fiber diet and bulk-forming agents

(2) Diarrhea predominant: antidiarrheals, antispasmodics

b. Drug therapy should be reserved for patients who do not respond to the above measures. It is usually initiated by the primary care provider and may include one or more of the following:

(1) Antidiarrheal agents

(2) Antispasmodic (anticholinergic) agents

(3) Anxiolytics

(4) Antidepressants

(5) A number of drugs, including 5-hydroxytryptamine (5-HT) (serotonin) 3 receptor antagonists and 5-HT 4 receptor agonists, have been removed from the market because of severe adverse effects.

H. Colonic diverticular disease

1. Diverticula are saclike herniations of colonic mucosa and submucosa through the muscularis; they may penetrate through areas of potential weakness in the bowel wall, such as where wall is penetrated by blood vessels.

a. Associated with a diet low in fiber and high in refined carbohydrates; promotes less bulky stools, increased GI transit time, and increased intracolonic pressure.

b. Although the location of diverticula may vary, most are confined to the sigmoid colon (left lower quadrant).

2. The frequency of diverticular disease is directly correlated with age, especially if the diet is low in fiber and roughage.

a. Frequency of diverticulosis increases with age: incidence is 10% in those <40 years old and as high as 70% in those 80 years old.

b. Most patients with diverticulosis remain asymptomatic.

c. Massive, painless lower GI bleeding in the elderly is one clinical presentation of diverticulosis. Occurs in up to 5% of patients with diverticular disease yet is the cause of 40% of lower GI bleeding.

d. In most instances, the bleeding stops spontaneously.

3. **Uncomplicated diverticulitis**

   a. **Only the pericolonic fat is inflamed.**

   b. **Pathophysiology: fecal material in the neck of a diverticulum hardens → fecalith → fecalith erodes the mucosa or compromises its blood supply → ↑ susceptibility to bacterial invasion → inflammation**

   c. **Clinical presentation is usually left lower quadrant pain with associated tenderness on examination. May also have suprapubic tenderness to palpation. Peritoneal signs are absent.**

      (1) **Patient usually appears well, lacking systemic toxicity.**

      (2) **Anorexia, nausea, and vomiting are sometimes present.**

      (3) **Fecal guaiac testing may be positive.**

      (4) **Ancillary testing primarily used to exclude complicated diverticulitis.**

      (5) **Caution in elderly and immunocompromised, because abdominal examinations and systemic response may be unreliable.**

      (6) **Patients are likely stable for outpatient therapy.**

4. **Complicated diverticulitis**

   a. **Process has advanced such that microperforation, abscess, or phlegmon has occurred. Fistula formation may occur, and 2% have free perforation.**

   b. **Physical examination is likely to show peritoneal signs with signs of systemic toxicity.**

   c. **Leukocytosis and sepsis may also occur.**

   d. **Abdominal CT is required to determine extent of pathology and identify complicating factors such as fistula, perforation, abscess.**

   e. Diagnostic evaluation

      (1) **CT of the abdomen is recommended as initial radiologic examination.**

         (a) **Helps establish the diagnosis when it is unclear**

         (b) **Helps establish extent of disease and complications**

         (c) **Excludes other etiologies**

         (d) **No risk of perforation**

      (2) **Colonoscopy is usually avoided when acute because of risk of perforation.**

      (3) **Barium enema is not done in acute stage.**

5. Management

   a. Uncomplicated diverticulitis: may be treated on outpatient basis

      (1) **High-fiber diet prevents recurrence for 5 years in 70% of patients.**

      (2) **Antibiotics to cover anaerobes and gram-negative organisms.**

         (a) **TMP-SMX + metronidazole *or***

         (b) **Ciprofloxacin + metronidazole *or***

         (c) **Amoxicillin-clavulanate for 10 days**

      (3) **Analgesics**

b. Complicated diverticulitis
   (1) IV fluids and broad-spectrum antibiotics to provide both aerobic and anaerobic bacterial coverage
      (a) Ciprofloxacin + metronidazole
      (b) Ticarcillin-clavulanate
      (c) Ampicillin-sulbactam
      (d) Imipenem for more severe cases
   (2) Nothing by mouth; bowel rest
   (3) Nasogastric suction if ileus or intestinal obstruction is present
   (4) Surgery required in only 10% of patients with acute disease
      (a) Small abscesses may be treated with antibiotics only; abscesses >4 cm may require percutaneous drainage.
      (b) Surgical consult for all with peritoneal signs or free air
   (5) Analgesia
      (a) Morphine can theoretically increase intraluminal pressure, but no studies confirm worsened outcome.
      (b) Morphine and hydromorphone preferred by many clinicians.

VI. ANORECTAL DISORDERS

A. Hemorrhoids

1. Definition
   a. Dilated venules of the hemorrhoidal plexuses
   b. Associated with chronic constipation, straining, increased intra-abdominal pressure, pregnancy, increased portal pressure, and a low-fiber diet
2. Types
   a. External hemorrhoids arise from below the dentate line (which separates the rectum from the anus) and are covered with well-innervated squamous epithelium. They are visible on external inspection.
   b. Internal hemorrhoids originate from above the dentate line and are covered by relatively insensitive rectal mucosa. Although not normally palpable on examination, they can be visualized with anoscopy at the 2, 5, and 9 o'clock positions and may visibly prolapse with the Valsalva maneuver.
3. Clinical presentation
   a. Patients with external hemorrhoids most commonly present with painful thrombosis. Examination reveals a tender mass at the external anal orifice.
   b. Patients with internal hemorrhoids usually present with painless bright-red bleeding that occurs in association with defecation. (Hemorrhoids are the most common cause of rectal bleeding). The blood covers the feces or drips into the toilet bowl and is usually small in volume.

4. Management
   a. Thrombosed external hemorrhoids
      (1) WASH regimen
      Warm water soaks
      Analgesics (mild oral analgesics)
      Stool softeners
      High-fiber diet
      (2) Topical steroids are of limited value and should be avoided.
      (3) Topical anesthetics may offer some relief, but their use is controversial.
      (4) If pain is severe and the thrombosis is acute (<48 hours): excision of clots under local anesthesia followed by warm sitz baths 6–12 hours after drainage, followed by WASH regimen
   b. Bleeding internal hemorrhoids
      (1) If bleeding is minor and resolves after defecation: increased dietary fiber and fluids, fecal softeners or bulk laxatives, and warm soaks
      (2) Persistent, refractory bleeding: IV fluid resuscitation (as needed), rectal packing, and surgical therapy
   c. Surgical indications
      (1) Continued bleeding
      (2) Strangulation or incarceration
      (3) Intractable pain or severe itching

B. Anal fissure

1. Definition: linear tears of the squamous epithelium of the anal canal
   a. Most (90%) are located in the posterior midline; however, in women they are occasionally found in the anterior midline. Lesions found elsewhere should prompt consideration of an underlying disease process (eg, inflammatory bowel disease, neoplasm, tuberculosis) and referral for further evaluation. Chronic fissures may be accompanied by a sentinel pile (a swollen external tag of skin at the base of the fissure) and are often confused with an external hemorrhoid.
   b. The most frequent anorectal disorders affecting infants and children, the most common cause of rectal bleeding in the first year of life, and the most common cause of painful rectal bleeding in adults
2. Etiology: usually produced by the passage of large, hard feces but may also be seen with severe diarrhea
3. Clinical presentation: The patient has a history of constipation and complains of severe pain with defecation that lingers for several hours before resolving. The pain is often so severe that the patient avoids defecating and is unwilling to have a digital rectal examination. Scant associated hematochezia is common. Inspection of the anus reveals the classic linear ulcer in the posterior midline.
4. Management
   a. Instruct the patient to do the WASH regimen:
      (1) Take warm 104°F (40°C) sitz baths 3 or 4 times per day (relaxes the sphincter).
      (2) Use topical nifedipine gel or nitroglycerin ointment to relax anal sphincter tone. Sphincter spasm often accompanies fissures and leads to cycle of tearing, spasm, increase in tear, worsening spasm.

(3) Use bulk laxatives and fecal softeners.

(4) Eat a high-fiber diet (prevents stricture formation).

(5) Use lidocaine ointment for pain relief and to facilitate healing.

b. Anal sphincterotomy (usually performed by colorectal surgeon) is surgical procedure of choice for refractory cases.

C. Perirectal (anorectal) abscesses

1. Tend to occur in any of the potential spaces near the anus or rectum (perianal, ischiorectal, supralevator, and intersphincteric)
2. Etiology: most anorectal abscesses result from obstruction of mucusproducing glands at the base of the anal crypts. Other causes include inflammatory bowel disease, cancer, radiation injury, trauma, tuberculosis, and lymphogranuloma venereum. The resulting infections are frequently polymicrobial and involve the colonic flora.
3. Perianal abscesses are the most common.
   a. Located outside the anal verge
   b. Patient complains of painful perianal mass.
   c. Likely afebrile with normal WBC count
   d. May be incised in the emergency department
4. Ischiorectal abscess
   a. Form outside the sphincter muscles and below levator ani
   b. Patient likely complains of buttock pain.
   c. Fever and increased WBC count are likely.
   d. Almost all require drainage in the operating room, because appearance maybe misleading.
5. Intersphincteric abscess
   a. Deep to anal sphincter and below levator ani
   b. Patient complaint of constant rectal pressure and increased pain with defecation and sitting.
   c. Rectal examination shows painful mass occasionally with purulent drainage.
   d. Inguinal adenopathy common
   e. Require drainage in the operating room
6. Supralevator abscess
   a. Perianal and buttock pain, often associated with fever
   b. External evidence of infection is lacking. Rectal examination may show tender mass.
   c. Surgical drainage in operating room required.
7. Management: anorectal abscesses require early and extensive drainage.
   a. Simple perianal abscesses may be drained in the emergency department under local anesthesia.
   b. All other perirectal abscesses need to be drained in the operating room.
   c. Routine use of antibiotics is not warranted; they should be reserved for immunocompromised patients, patients with diabetes or valvular heart disease, and patients with associated cellulitis.
8. Complication: fistula formation is a common sequela.

D. Miscellaneous anorectal disorders

1. Fistula-in-ano
   a. An abnormal tract between the anal canal and skin that is lined with granulation tissue
   b. Most commonly occurs as a complication of a perianal or ischiorectal abscess; may also be associated with ulcerative colitis, Crohn disease, tuberculosis, radiation, and cancer of the anus or rectum
   c. If the tract remains open, there is a persistent, purulent, blood-stained discharge; if the tract becomes blocked (most common), an abscess may be the only physical finding.
   d. Treatment is surgical excision.
2. Rectal prolapse (procidentia)
   a. Types
      (1) Mucosal prolapse (seen primarily in young children and in association with internal hemorrhoids)
      (2) Prolapse involving all layers of the rectum
      (3) Intussusception of the upper rectum into the lower rectum with the apex protruding through the anus
   b. Complete rectal prolapse occurs in the very young and the very old (particularly older women).
   c. Rectal prolapse in children should prompt consideration of cystic fibrosis.
   d. Clinical presentation: patients typically complain of a painless anal mass noticed after straining, coughing, or defecation.
   e. Management
      (1) Reduction can be accomplished in almost all patients, although recurrence (particularly in older patients) is likely.
      (2) In the presence of vascular compromise, emergency reduction is mandatory.
      (3) After reduction of the prolapse, patients should be placed on fecal softeners and referred for further evaluation (to uncover any underlying pathology) and potential surgical correction (commonly required in the elderly).
3. Pilonidal sinus (abscess)
   a. Occurs in the midline (at the superior edge of the buttock crease) and is more common in men than women
   b. The sinus is formed when an ingrowing hair penetrates the skin and induces a foreign body granuloma reaction.
   c. A single opening at the base of the spine with hair protruding is the most common physical finding.
   d. When the sinus becomes plugged and cannot drain → abscess
   e. Management
      (1) Surgical incision longitudinal and lateral to the sacral midline with drainage of purulent material
      (2) Refer for definitive surgical care.
      (3) Antibiotics are indicated if there is an associated cellulitis or the patient is immunocompromised.
4. Anorectal tumors
   a. Classification: based on their virulence, these tumors have been arranged into two groups with distinct anatomic locations.

(1) Anal canal neoplasms (found proximal to the dentate line)
(a) Represent 80% of anorectal tumors
(b) Have a high-grade malignant potential and metastasize early; prognosis is pc
(c) Tumors occurring in this region
i. Adenocarcinoma (common)
ii. Mucoepidermoid carcinoma
iii. Malignant melanoma
iv. Kaposi sarcoma
v. Squamous cell carcinoma
vi. Basaloid carcinoma
vii. Villous adenoma
(2) Anal margin neoplasms (found distal to the dentate line)
(a) Represent 20% of anorectal tumors
(b) Have a low-grade malignant potential, slow to metastasize, good prognosis
(c) Tumors occurring in this region include:
i. Basal cell carcinoma
ii. Squamous cell carcinoma
iii. Bowen disease
iv. Extramammary Paget disease
v. Giant solitary trichoepithelioma

b. Clinical presentation: pruritus, rectal bleeding, pain, weight loss, anorexia, constipation, decrease in fecal caliber, tenesmus, and obstruction
c. Management: prompt surgical referral

5. Rectal foreign bodies
a. Diagnostic evaluation
(1) Abdominal radiographs should be obtained to ascertain the location, size, shape and number of objects present as well as to exclude the presence of free air.
(2) Multiple views may be needed.
b. Management
(1) If there is clinical or radiographic evidence of free air, the patient should be prepared for surgery and an immediate surgical consult obtained.
(2) After removal of the foreign body, the patient should be observed for 12 hours, a radiograph studies and sigmoidoscopy performed.
c. Complications
(1) Perforation is the most frequent; it results from the foreign body itself or attempts remove it.
(2) Mucosal tears can also occur.

VII. DIARRHEA AND FOOD POISONING

A. General information

1. The vast majority of patients in the emergency department suspected of having "gastroenteritis" have a benign, self- limited disease. Evaluation should distinguish between acute or chronic diarrhea as well as the presence of invasive or noninvasive organisms.
2. Acute gastroenteritis is defined as symptoms being present for <2 weeks. The cause is either viral or bacterial in most cases.
3. Chronic gastroenteritis (symptoms for >2 weeks) suggests an alternative cause such as intestinal parasites or other disorders.
4. Invasive gastroenteritis is associated with fever, abdominal pain, blood in the feces, and tenesmus. Presence of these symptoms suggests a bacterial cause, and testing for specific organisms may be warranted.
5. Noninvasive disease is suggested by the absence of the above symptoms. The feces are not bloody in most cases. Suggests a viral cause or pre-formed toxin, and testing is rarely warranted.
6. If tested, the presence of fecal blood and leukocytes helps distinguish invasive from noninvasive.

B. Viral diarrheal diseases (most acute episodes of diarrhea)

1. Although many viruses can cause gastroenteritis, two groups have become most common over the last 10 years: reovirus-like agents such as rotavirus and the noroviruses, which include Norwalk virus.
   a. Rotavirus infection
      (1) Outbreaks are usually sporadic, usually in winter months.
      (2) Primarily in children 6–24 months old
      (3) Incubation period of 1–3 days
      (4) Clinical presentation
         (a) Onset usually abrupt with vomiting, low-grade fever, and watery diarrhea. Vomiting usually lasts 1–1.5 days and resolves spontaneously. Diarrhea may last 4–7 days.
         (b) Abdominal pain not common
      (5) Diagnostic evaluation: diagnosis is confirmed by detection of antigens in the feces by latex agglutination, PCR, or ELISA. However, testing is not usually required.
      (6) Not uncommonly, children become dehydrated and require IV hydration and hospitalization. The disease is an uncommon cause of mortality in the United States.
      (7) A vaccine, introduced in 2006, is given to babies at 2, 4, and 6 months of age.
   b. Norwalk virus
      (1) Up to 80% of diarrheal illness in industrialized countries
      (2) Can be transmitted person to person by fecal-oral route
      (3) Water and food-borne outbreaks common; up to 50% of food-borne outbreaks caused by norovirus
         (a) Stored water on cruise ships a common source
         (b) Shellfish, food handlers; raw oysters implicated

(4) In January 2013, the CDC reported a new strain, GII.4 Sydney, which appears to cause more severe disease and results in more hospitalizations and deaths.

(5) Clinical presentation

(a) After an incubation period of 1–2 days, patients typically have an abrupt onset of nausea, nonbloody watery diarrhea, abdominal cramps, and low-grade feve Vomiting is not prominent.

(b) There is no upper or lower respiratory tract involvement.

(c) Symptoms last for 1–2 days.

(6) Management: disease is self-limited and requires only supportive, symptomatic care

C. Bacterial diarrheal diseases (20% of acute diarrheal cases)

1. Classification

a. Invasive bacteria act primarily on the large bowel and produce diarrhea by damaging cell membranes and eliciting an inflammatory response → blood and mucus in the feces

(1) *Campylobacter*

(2) *Salmonella*

(3) *Shigella*

(4) *Vibrio parahaemolyticus*

(5) *Vibrio vulnificus*

(6) *Yersinia enterocolitica*

(7) Enteroinvasive *E coli*

(8) Enterohemorrhagic *E coli* serotype O157:H7

(9) *Clostridium difficile*

b. Enterohemorrhagic *E coli* serotype O157:H7 and *C difficile* are actually toxin-producing bacteria that induce diarrhea by elaborating a cytopathic toxin that destroys the intestinal wall. They are grouped with the invasive bacteria, because the clinical syndrome they produce most closely resembles that produced by these organisms.

c. Enterotoxin-producing bacteria release a toxin that acts primarily in the small intestine and produces diarrhea by altering water and electrolyte transport in epithelial cells → profuse watery diarrhea

(1) *Staphylococcus aureus*

(2) *Bacillus cereus*

(3) Ciguatera fish poisoning

(4) Scombroid fish poisoning

(5) Enterotoxigenic *E coli*

(6) *Clostridium perfringens*

(7) *Aeromonas hydrophila*

(8) *Vibrio cholera*

2. Fecal leukocytes (a wet mount or methylene blue stain of the feces)

a. May be used as a screen for those patients who may benefit from antibiotic therapy

(1) Invasive bacterial pathogens producing fever, systemic illness, or bloody feces: *Salmonella, Shigella, Campylobacter,* and *E coli* O157:H7

(2) Pseudomembranous enterocolitis

(3) Amebiasis
(4) Inflammatory bowel disease (Crohn disease and ulcerative colitis)

Table 7: Fecal Leukocytes in Diarrheal Syndromes

Fecal Leukocytes Present	Fecal Leukocytes Absent
Campylobacter	Rotavirus
Salmonella	Norwalk virus
Shigella	Enteric type adenovirus
Vibrio parahaemolyticus	*Staphylococcus aureus*
Vibrio vulnificus	*Bacillus cereus*
Yersinia	Ciguatera fish poisoning
Amebiasis	Scombroid fish poisoning
Enteroinvasive *E coli*	Enterotoxigenic *E coli*
Enterohemorrhagic *E coli*	*Clostridium perfringens*
Clostridium difficile	*Aeromonas hydrophila*
Ulcerative colitis	*Vibrio cholera*
Crohn disease	*Giardia lamblia*

b. Although treatment based on the presence or absence of fecal leukocytes is not 100% accurate, it is a good place to start, and is one of the few test results for diarrheal illnesses that is immediately available. Presence of fecal leukocytes (>5 WBCs per high-power field) has an LR of 4.5 for the presence of an invasive pathogen.

3. Traveler's diarrhea
   a. Definition: syndrome acquired by travelers who eat food or water that is fecally contaminated
   b. Risk factors
      (1) Travel to developing tropical regions (Mexico, Latin America, southern Asia, Africa) where sanitation is poor
      (2) Ingestion of food prepared by street vendors
      (3) Ingestion of certain unsafe (often contaminated) items such as tap water, ice cubes, unpasteurized dairy products, raw vegetables, unpeeled fruit, and raw or undercooked seafood or meat
   c. Most cases (70%–75%) are caused by bacteria, of which enterotoxigenic *E coli* is the most commonly involved pathogen; it is responsible for up to 50% of cases worldwide.
   d. Clinical presentation: symptoms often begin abruptly and consist of watery diarrhea and abdominal cramps; other associated symptoms may be present and vary with the particular etiologic agent involved.
   e. Antibiotic prophylaxis
      (1) Not recommended for healthy individuals because of the associated risks (eg, emergence of resistant organisms, allergic reactions, photosensitivity reactions, etc)
      (2) Bismuth subsalicylate 2 tablets 4 times a day and strict adherence to preventive methods (avoiding the above) recommended by the CDC.
      (3) Initiation of treatment at the onset of symptoms is preferred.

f. Management

(1) Oral rehydration with electrolyte replacement such as found in sports drinks, caffeine-free soft drinks, or diluted fruit juice effectively maintains fluid and electrolyte balance in most patients.

(2) Antibiotics

(a) The CDC recommends a fluoroquinolone such as ciprofloxacin or norfloxacir for 3–5 days.

(b) Rifixamin also approved for treatment at onset of symptoms.

(c) TMP-SMX no longer recommend because of resistance.

(3) Antimotility agents such as loperamide provide rapid symptomatic relief but shou not be continued for >2 days and should not be used in patients who have fevers bloody diarrheas, because these may increase severity and prolong symptoms.

(4) Bismuth subsalicylate is an antisecretory agent that is also effective in reducing th number of bowel movements and the duration of illness. It works by decreasing tl outpouring of fluids by the mucosa of the small intestine.

4. Specific bacterial infections

a. ***E coli:*** **several strains have been shown to be diarrheagenic, two of which are discussed below. These strains differ in terms of their virulence, epidemiology, and clinical characteristics.**

(1) Enterotoxigenic *E coli*

(a) Most common cause of traveler's diarrhea worldwide

(b) Acquired by ingesting fecally contaminated food and water

(c) Causes diarrhea by producing both heat-labile and heat-stable toxins

(d) Clinical presentation

i. Incubation period 1–4 days

ii. Symptoms usually begin suddenly and consist of watery diarrhea associat with abdominal cramping.

(e) Management

i. Most cases resolve in 2–3 days with supportive measures alone.

ii. The use of antibiotics and antimotility agents (see traveler's diarrhea, above) can shorten the duration of this syndrome.

(2) Shiga toxin–producing *E coli* (STEC), also known as enterohemorrhagic *E coli*

(a) Serotype O157:H7 is most common isolate in the United States.

(b) Transmission occurs by ingesting small amounts of human or animal feces. Person-to-person spread is also possible. Exposure to cattle can also transmi disease.

(c) Outbreaks commonly occur due to improperly cooked ground beef, fruits, or vegetables. High-risk foods are raw milk, soft cheeses made from unpasteurized milk, unpasteurized apple cider, and nondisinfected water.

(d) Outbreaks are associated with ingestion of contaminated, undercooked ground beef, seed sprouts, or unpasteurized milk; however, person-to-perso spread also occurs.

(e) Most common in children, the elderly, and post-gastrectomy patients.

(f) This organism produces Shiga toxins that are cytotoxic to the intestinal vascular endothelium.

(g) Clinical presentation

i. Incubation period 3–4 days on average

ii. Patient presents with diarrhea, crampy abdominal pain, and vomiting. The cramps can be severe enough to masquerade as an acute abdomen. The diarrhea is watery early on but eventually becomes bloody. Fever is absent or low grade. WBCs are present on the wet mount but are few in number.

(h) Diagnostic evaluation: fecal testing for Shiga toxin along with cultures for *E coli* O157:H7

(i) Management

i. Antibiotic therapy is contraindicated; antibiotics have not been shown to decrease the duration of symptoms, and they may increase the risk of hemolytic uremia syndrome.

ii. Antidiarrheal agents may also increase the risk of hemolytic uremia syndrome.

iii. Most cases resolve in 7–10 days with supportive measures.

(j) Complications

i. Hemolytic uremic syndrome

- Occurs in about 8% of children, of which 5%–10% of cases are fatal
- Occurs in nearly 40% of elderly patients who acquire STEC in a nursing-home outbreak; up to 80% fatality rate

ii. Thrombotic thrombocytopenic purpura develops in ~2% of patients, usually immunocompromised.

b. *Shigella* (a common cause of bacterial diarrhea)

(1) Shigellosis (bacillary dysentery) occurs worldwide but is especially common in countries where adequate sanitary facilities are lacking.

(2) The CDC estimates an annual incidence in the United States of 4 cases per 100,000 persons.

(3) Common in "contained" populations, eg, nursing homes, prisons, schools, day care facilities; outbreaks have been reported in water parks, swimming pools, etc.

(4) *Shigella sonnei* causes ~75% of cases in the United States; *Shigella flexneri* causes most of the rest.

(5) Clinical presentation

(a) Incubation period is 24–48 hours after exposure to even a very small inoculum.

(b) Mild, watery diarrhea with scant systemic symptoms may be seen in most early disease. Dysentery can then ensue with fevers, crampy abdominal pain, grossly bloody diarrhea, vomiting, headaches, and myalgia. Profound dehydration may develop, especially in infants and the elderly.

(c) Convulsions may occur in young children, usually those <2 years old. The mechanism is unclear but may be due to rate of rise of the fever or metabolic alterations.

(d) Disease is generally self-limited and resolves within a week. However, relapse is common without antibiotic treatment.

(6) Diagnostic evaluation

(a) CBC: leukocytosis with a marked left shift; an absolute band count >800 is suggestive of shigellosis.

(b) A wet mount of the feces with methylene blue demonstrates numerous fecal leukocytes.

(c) Fecal cultures are positive in most cases, particularly if obtained within the first few days of illness.

(7) Management

(a) Fluid resuscitation or maintenance of hydration is of paramount importance.

(b) Antibiotics decrease the duration of illness and excretion of Shigella organisms in the feces. They are recommended in patients with dysentery, in the very young and very old, and in institutional outbreaks. Management should continue for 3 days in immunocompetent patients and for 10 days in immunocompromised.

i. Quinolones (ciprofloxacin, norfloxacin, ofloxacin) are the drugs of first choice, but they are contraindicated in pregnancy and in children <17 years old.

ii. Ceftriaxone may be useful.

iii. Ampicillin and TMP-SMX are not effective and are no longer considered viable treatment options unless fecal culture indicates effectiveness.

(c) Antimotility drugs should be avoided, because they can prolong duration of disease.

(8) Complications

(a) Dehydration secondary to profuse diarrhea

(b) Arthralgias

(c) Reiter syndrome (nongonococcal urethritis, polyarthritis, conjunctivitis)

(d) Hemolytic uremic syndrome

(e) Febrile seizures (common in children <3 years old)

(f) Pneumonitis

c. *Salmonella*

(1) Second most common cause of food-borne illness in the United States

(2) Many serotypes of *Salmonella* infect animals as well as people. *S typhi*, the cause of typhoid fever, is the most virulent serotype, whereas *S typhimurium* is the most common.

(3) **Nearly all cases of *Salmonella* infection are acquired by ingesting contaminated food or drink. Poultry and beef are the most common source of contamination. Unpasteurized milk, eggs, and fish are other sources. Ingestion of raw eggs is a common exposure.**

(4) Infection can result in a variety of clinical syndromes, including gastroenteritis, septicemia (enteric fever), typhoid (enteric) fever, and asymptomatic carrier state.

(5) High-risk groups

(a) Children (<5 years old) and the elderly

(b) Immunosuppressed patients

(c) Splenectomized patients

(d) Patients with hemolytic anemias (including sickle cell anemia)

(6) Clinical presentation

(a) *Salmonella* gastroenteritis: patients present with nausea, vomiting, diarrhea, fever, and crampy abdominal pain 8–48 hours after ingestion of contaminated

food. The feces are watery, foul, brownish green, and contain blood, leukocytes, and mucus. The disease course is generally selflimited with symptoms lasting only a few days.

(b) Septicemia develops in 10%–15% of patients and is indistinguishable from other causes of sepsis. It is most common in infants, the immunosuppressed, and those with hemoglobinopathies. Focal infections generally involve sites adjacent to the bowel (such as the gallbladder), but seeding of the brain and other (more distant) organs may occur.

(c) Typhoid (enteric) fever is characterized by septicemia with a protracted toxic course.

i. An incubation period of 1–2 weeks caused by *S typhi*

ii. Intractable fever

iii. Relative bradycardia; bradycardia despite high fever

iv. Absence of diarrhea

v. Crampy abdominal pain

vi. Maculopapular skin lesions ("rose spots")

(d) Asymptomatic carrier state

(7) Diagnostic evaluation

(a) Stained fecal smear reveals numerous WBCs and, occasionally, occult blood.

(b) Fecal cultures confirm the diagnosis.

(c) Blood cultures are reserved for toxic patients; they are the best method for diagnosing typhoid fever.

(8) Management

(a) *Salmonella* gastroenteritis

i. Uncomplicated cases in otherwise healthy patients should be treated with fluid replacement alone; these patients do not require antibiotic therapy.

ii. Patients with serious underlying illness or immunosuppression should receive antimicrobial therapy, although antibiotics may prolong the duration of shedding. A fluoroquinolone (eg, ciprofloxacin, norfloxacin) is the drug of choice. TMP-SMX is also effective.

(b) Septicemia or typhoid fever (enteric fever): ceftriaxone is the agent of choice; alternatives include ciprofloxacin, chloramphenicol, and ampicillin.

(c) Antidiarrheal agents are contraindicated, because they prolong the illness and increase the incidence of bacteremia and the carrier state.

d. *Campylobacter* enteritis

(1) *Campylobacter* infection is the most common cause of bacterial diarrhea.

(2) It is transmitted by ingestion of fecally contaminated water or food (particularly poultry) or by direct contact with feces of infected animals or people; it produces disease by directly invading the colonic epithelium.

(3) Clinical presentation

(a) Incidence of infection is greater in young children.

(b) Incubation period is 2–5 days.

(c) Onset of disease is usually rapid and consists of fever and crampy, abdominal pain. Malaise, myalgia, and headache are common. Abdominal pain may be severe enough to mimic an acute abdomen.

(d) Diarrhea usually begins 1–2 days after the fever and pain. It is frequently loos and bile colored, subsequently turning watery and bloody. Gross blood is see in 60% of cases.

(4) Diagnostic evaluation

(a) Wet mount shows blood and fecal leukocytes.

(b) Fecal culture confirms the diagnosis.

(5) Management

(a) Antimotility agents should be avoided.

(b) Patients with mild disease do not require treatment other than supportive care. Empiric antibiotics are not recommended.

(c) Patients who are still symptomatic when culture results are returned may be treated with erythromycin or azithromycin. Ciprofloxacin in no longer recommended because of widespread resistance.

(6) Complications

(a) Reiter syndrome

(b) Hemolytic uremic syndrome

(c) Guillain-Barré syndrome (a late sequela)

e. *Yersinia* enterocolitis

(1) Infection results from ingestion of contaminated food or drink and is most comm in childhood.

(2) Clinical presentation

(a) Patient presents with fever and diarrhea 2–6 days after exposure that is accompanied by severe, cramping abdominal pain that may last up to 2 week

(b) Dysentery (bloody diarrhea) may be present in up to 25% of patients.

(c) On physical examination, the abdomen is tender and, if the organism has caused mesenteric adenitis or terminal ileitis (which it sometimes does), you may think the patient has appendicitis.

(d) Recent exposure to domestic, farm, or wild animals can be a significant diagnostic clue.

(e) Postinfectious manifestations (erythema nodosum, polyarthritis) sometimes occur, particularly in adults.

(3) Diagnostic evaluation

(a) Wet mount of the feces shows fecal leukocytes and occasionally RBCs.

(b) Fecal culture (special techniques are required) confirms the diagnosis.

(4) Management

(a) Mild to moderate infections: supportive therapy; however, *Yersinia* takes a lon time to isolate and identify.

(b) If antibiotics are started pending results, TMP-SMX is the antibiotic of choice. Quinolones are also effective.

(c) Antimotility drugs should be avoided.

f. *Clostridium perfringens*

(1) A common cause of food poisoning; relatively large outbreaks are typical.

(2) Food poisoning occurs when food contaminated with the live organism containir type A heat-resistant spores are ingested. Typically, the food (meat or poultry, gravies, steam-table meats) was prepared earlier and allowed to cool. *C perfringe*

multiplies and is then ingested. In the gut, the organism produces an enterotoxin → diarrhea and abdominal cramps

(3) Clinical presentation

(a) Incubation period is generally 1–5 hours.

(b) Patient presents with significant vomiting with abdominal cramping. Diarrhea is present in 25%.

(c) Fever, headache, and chills may also be present but are not prominent features.

(d) No WBCs are seen on a wet mount of the feces.

(4) Management

(a) This is a self-limited disease, with symptoms resolving in 10–12 hours.

(b) Only supportive treatment is indicated.

g. *Staphylococcus aureus* (the most common cause of food poisoning)

(1) Illness results from ingestion of a heat-stable enterotoxin.

(a) Cooking at a temperature >140°F (60°C) will kill bacteria but not destroy the toxin.

(b) Most protein-rich foods (eg, ham, eggs, mayonnaise, potato salad) support growth of staphylococci.

(c) Contaminated food has no abnormal odor or taste.

(2) Large outbreaks are common; the attack rate is >75%.

(3) Clinical presentation

(a) Incubation period is 1–6 hours, followed by abrupt onset of abdominal cramping and violent vomiting. Diarrhea may or may not occur.

(b) Usual scenario: patient went to a barbecue or buffet about 6 hours before arrival where he or she had eaten some potato salad or sliced ham that had been sitting at room temperature for several hours.

(4) Treatment is supportive; resolves in 6–10 hours.

h. *Bacillus cereus*

(1) Two clinical syndromes

(a) An emetic syndrome caused by a heat-stable enterotoxin

i. Begins 1–6 hours after ingestion of contaminated food. Fried rice is a common source; beef, poultry and milk have been implicated.

ii. Symptoms are indistinguishable from those of staphylococcal poisoning. Abrupt onset of vomiting and crampy abdominal pain. Up to 25% have diarrhea.

iii. Treatment is supportive; resolves in ≤10 hours.

(b) A diarrheal syndrome caused by a heat-labile enterotoxin

i. Begins 6–24 hours after ingestion of contaminated meal (usually meat or vegetables)

ii. Syndrome is similar in presentation to that of *Clostridium* poisoning. Symptoms include diarrhea and abdominal cramps. Vomiting is present in 20%.

(2) Diagnosis can be confirmed by isolation of >105 colonies of *B cereus* from the food source. However, because of the extremely short nature of the disease, this is rarely done.

(3) Treatment is supportive; resolves in 10–24 hours.

i. *Aeromonas hydrophila*
   (1) Usually acquired by drinking untreated water from a well or spring
   (2) Most commonly affects children and immunocompromised patients
   (3) Clinical presentation
      (a) Watery diarrhea, crampy abdominal pain, vomiting (25%), fever (50%)
      (b) History of drinking from wells or fresh springs increases likelihood.
   (4) Diagnostic evaluation
      (a) Confirmed by fecal culture; however, routine culture does not test for *Aeromonas*, so it must be specifically requested.
      (b) Fecal leukocytes and occult blood are usually absent. However, some patients present with symptoms suggesting colitis and have fecal leukocytes and blood; this may mimic Crohn disease.
   (5) Management
      (a) Supportive measures will suffice in most patients, because disease is usually self-limited.
      (b) Antibiotic therapy with one of the following agents:
         i. TMP-SMX has shown good activity.
         ii. Fluoroquinolones are also effective.
      (c) Without antimicrobial therapy, the diarrhea generally persists for ≥2 weeks.

j. *Vibrio cholera*
   (1) Transmitted by ingestion of contaminated water or seafood (especially raw oysters), which produces an enterotoxin-mediated diarrheal illness (cholera).
   (2) Large epidemics related to ingestion fecally contaminated water supplies.
   (3) Clinical presentation
      (a) Copious amounts of watery diarrhea ("rice water" feces) is the hallmark of clinical cholera and can lead to significant fluid and electrolyte imbalances.
      (b) Severe dehydration (loss of isotonic fluid from bowel)
      (c) Hyperchloremic acidosis (loss of bicarbonate in the feces)
      (d) Hypokalemia (loss of potassium in the feces)
      (e) After an incubation period of 2 days, the patient presents with copious watery diarrhea and abdominal distention. Vomiting may also be present.
   (4) Diagnostic evaluation
      (a) Diagnosis is confirmed by fecal culture.
      (b) Crystal VC® rapid diagnostic test can be used for rapid identification of two serotypes in epidemic situations to establish diagnosis.
   (5) Management
      (a) Fluids (oral or IV) should be the focus of therapy.
      (b) Antibiotic therapy in those with moderate to severe dehydration. Doxycycline is drug of choice in aduts; azithromycin is first choice in children and pregnant women.
      (c) Zinc is recommended for all patients to decrease duration and quantity of diarrhea.

k. *Vibrio parahaemolyticus*
   (1) Leading cause of seafood-associated gastroenteritis in the United States

(2) Invasive bacterial infection; other *Vibrio* species produce an enterotoxin to cause disease.

(3) Acquired by ingestion of raw or improperly prepared seafood (especially oysters, clams, shrimp, and crabs); exposure to brackish or salty water is potential cause as well.

(4) In temperate climates, this infection is usually confined to the summer months.

(5) Clinical presentation

(a) Symptoms begin after an average incubation period of 12 hours and range from mild gastroenteritis to explosive diarrhea with vomiting, cramps, and dysentery.

(b) Fever may also be present.

(c) Can also cause wound infection if open wound exposed to seawater

(6) Diagnostic evaluation

(a) Wet mount of the feces reveals numerous WBCs.

(b) Fecal culture requires special growth media, so laboratory must be notified.

(7) Management

(a) Symptomatic therapy is generally all that is needed, because the illness is self-limited and resolves in 2–3 days.

(b) Tetracycline or a quinolone may be given for severe infections (dysentery), but their efficacy remains unclear; they neither shorten the clinical course nor decrease the duration of shedding of the organism.

l. Scombroid fish poisoning

(1) The most commonly implicated fish is mahi mahi (blue dolphin or dolphin fish), but it is also common with ingestion of tuna, mackerel, and other dark-fleshed or red-muscled fish.

(2) Most cases are in Hawaii and Florida, although it can be seen anywhere "fresh fish" is flown in.

(3) Poisoning results from ingestion of heat-stable toxins with histamine-like properties produced by bacterial action on dark-meat fish; high levels of histamine correlate with the manifestations of the illness. Formation of the toxin is related to improper preservation and refrigeration of the fish. (This is not an allergic reaction.)

(4) Clinical presentation

(a) Patient presents with signs and symptoms of histamine intoxication 20–30 minutes after ingestion of the fish:

i. Facial flushing (resembles a sunburn and can extend over entire upper body)

ii. Throbbing headache

iii. Abdominal cramps

iv. Nausea, vomiting, and diarrhea

v. Palpitations

vi. Bronchospasm or hypotension (with severe toxicity)

(b) Historical clue: at time of ingestion, the patient noticed a sharp, metallic, bitter, or peppery taste to the fish.

(5) Diagnostic evaluation

(a) Diagnosis is based on clinical grounds with symptoms beginning shortly afte ingestion of the fish.

(b) If necessary, histamine levels can be checked on the suspect fish.

(6) Management

(a) Depending on severity of symptoms, H_1- and H_2-blockers should be administered IV or orally.

(b) Activated charcoal may be administered if a large amount of fish was ingest

(c) Additional therapy may be required.

i. Antiemetics (for nausea and vomiting)

ii. Albuterol and steroids (for bronchospasm)

iii. Epinephrine (for anaphylactoid reaction)

m. Ciguatera fish poisoning

(1) Most common cause of nonbacterial fish-associated food poisoning in the United States

(2) Results from ingestion of ciguatoxin, a tasteless, odorless, heat-stable neurotoxi that accumulates in the flesh of certain carnivorous tropical and semitropical coral-reef fish when a particular dinoflagellate is present in their food chain during the late spring and summer months. The most commonly affected species are grouper, snapper, barracuda, king fish, and jack. Because the toxin accumulates in the flesh of these fish, the bigger and older the fish, the more likely that it carries the toxin.

(3) Clinical presentation

(a) Ciguatoxin affects the sodium channels, so effects are most pronounced in neurvous, cardiac, and GI tissues.

(b) Incubation period is usually 2–6 hours but ranges from 15 minutes to 24 hours.

(c) GI symptoms last 1–2 days and include abdominal pain, nausea, vomiting, a diarrhea.

(d) Neurologic symptoms occur within hours up to 3 days and include painful paresthesias, circumoral and throat paresthesias, weakness, and coma. Paralysis of respiratory muscles has been reported. Pruritus is a common complaint. Neurosensory symptoms may persist for months and are exacerbated by ingestion of alcohol.

(e) Pathognomonic "hot-cold reversal" likely represents intense dysesthesia experienced with cold stimuli rather than true reversal of temperature perception.

(f) Cardiovascular symptoms occur least frequently but may include symptoma bradycardia.

(4) Management

(a) Supportive measures (IV fluids, antiemetics, analgesics)

(b) H_1- and H_2-blockers may help with pruritus. Amitriptyline may be useful for pruritus and dysesthesia.

(c) IV mannitol is no longer considered an effective therapy and is not recommended.

(d) Absolute abstinence from alcohol, seafood, and nuts until all symptoms have resolved; continued abstinence is recommended for 3–6 months after exposure.

5. Summary recommendations for gastroenteritis
   a. Antibiotics should be limited to patients who are significantly ill or toxic in appearance that are believed (based on clinical grounds) to have an invasive or infectious diarrhea.
   b. Early administration of antibiotics to these patients decreases the intensity and duration of symptoms, prevents systemic and suppurative sequelae, and reduces spread of infection.
   c. Unless contraindications exist (eg, pregnancy, children <17 years old), fluoroquinolones are the agents of choice; they are effective against all the common bacterial causes of infectious diarrhea and have few adverse effects. Acceptable regimens include:
      (1) Ciprofloxacin 500 mg orally bid for 3–5 days
      (2) Norfloxacin 400 mg orally bid for 3–5 days
      (3) Azithromycin if *Campylobacter* is suspected.

D. Parasitic GI infections: all patients with GI symptoms lasting >7 days should be tested for parasites.
   1. *Entamoeba histolytica*
      a. Protozoa with cystic and trophozoite phases
      b. Transmission usually occurs through ingestion of cysts (the infective phase) in water or food contaminated with feces. Cysts are stable in the environment and passed via fecal-oral transmission. Infection occurs as the cysts change into a trophozoite phase in the terminal ileum or colon. This is the invasive form, which causes symptoms.
      c. Trophozoites can invade the mucosal barrier of the colon and produce bloody diarrhea and colitis, which may resemble inflammatory bowel disease. It is estimated that only 10%–20% of those exposed develop disease symptoms.
      d. Risk factors
         (1) People who have traveled to tropical areas with poor sanitation; St. Petersburg, Russia, has had an ongoing outbreak.
         (2) Immigrants from tropical areas with poor sanitation
         (3) People who live in institutions with poor sanitation
         (4) Native Americans living on reservations
         (5) Men who have sex with other men
      e. Clinical presentation
         (1) Chronic amoebic colitis is the usual manifestation.
            (a) Gradual onset with intermittent diarrhea
            (b) Foul-smelling feces with bloody mucus
            (c) Mild abdominal cramping and flatulence
         (2) Acute amoebic dysentery
            (a) 1 week up to a year after exposure
            (b) Sudden onset of severe abdominal cramping
            (c) Profuse bloody diarrhea
         (3) Amoebic abscess

(a) Liver (most common)
(b) Fever, right upper quadrant pain and tenderness, weight loss
(c) Usually diagnosed by ultrasound or CT

f. Diagnostic evaluation
(1) Fecal studies
(a) Trophozoites from single fecal specimen only 33% sensitive; examination of three samples within 10 days is 90% sensitive.
(b) Fecal leukocytes may be present in small numbers.
(c) Fecal examination in patients with extraintestinal amebiasis is rarely helpful, because organisms are found in <20% of these patients.
(2) Sigmoidoscopy with rectal biopsy can be helpful when fecal examination is negative.
(3) World Health Organization recommends antigen-specific fecal or serologic testing be performed because of greater specificity.
(4) Eosinophilia is absent.

g. Management
(1) Asymptomatic carriers should be treated with a luminal agent such as iodoquinol to eradicate infection; prevents development of invasive disease and prevents shedding of cysts.
(2) Mild to moderate intestinal illness: metronidazole (or tetracycline) plus a luminal agent such as iodoquinol or paromomycin

2. Giardiasis
a. Causative organism is *Giardia lamblia* (also known as *Giardia intestinalis*); most frequently diagnosed parasitic disease in the United States and travelers with chronic diarrhea.
b. Transmitted by cysts via the fecal-oral route.
(1) Found in soil, food, and water that have been contaminated with feces containing the parasite.
(2) The parasite is protected by an outer coat that allows it to survive outside the body for long periods of time. This also makes it resistant to disinfection by chlorine.
(3) Most common method of transmission is by drinking contaminated water, ponds, streams etc; often called "backpackers diarrhea."
(4) Beavers play a role in the spread of this disease by contaminating streams with cysts.
(5) Venereal transmission among homosexuals (by direct fecal-oral contamination) is also common.
c. Symptoms, if they occur, are produced by trophozoites and begin after an incubation period of 1–3 weeks; most patients, however, are asymptomatic.
d. Risk factors
(1) Homosexual men
(2) Immunocompromised patients (IgA deficiency)
(3) People who live in institutions
(4) Native Americans living on reservations
(5) Children attending daycare centers
(6) Travelers returning from underdeveloped countries (especially Russia) or a backpacking trip to Colorado
(7) Patients with decreased gastric acidity from any cause

e. Classic clinical scenario: The patient has recently returned from a back-packing trip in Colorado (or from travel to Russia) and presents with abdominal pain and feeling bloated and gaseous. Most common symptoms are explosive diarrhea, flatulence, colicky pain, and greasy, floating, foul-smelling feces.

f. Diagnostic evaluation

(1) Presence of trophozoites or cysts in the feces; several samples over separate days may be required.

(2) Fecal immunoassays are available that are more sensitive and specific. The CDC recommends using these tests to screen high-risk patients (eg, daycare), but ova and parasite examination of the feces is the primary means of diagnosis.

(3) Duodenal aspiration may need to be performed in asymptomatic patients and those with chronic symptoms.

(4) Eosinophilia is absent.

g. Management

(1) Asymptomatic patients who shed the organism should be treated only to prevent spread to high-risk household contacts.

(2) All asymptomatic patients should be treated.

(a) Metronidazole (drug of choice)

(b) Furazolidone (less effective than metronidazole but is available as a suspension and, for this reason, is often preferred in children)

(c) Tinidazole (a congener of metronidazole or albendazole)

h. Patients with recurrent or refractory giardiasis should be tested for immunoglobulin A deficiency.

3. *Cryptosporidium*

a. Intestinal protozoan coccidial parasite; causes disease with clinical features similar to those of *Isospora*.

b. Most common cause of chronic diarrhea in patients with AIDS

c. Transmitted by cysts from human or animal sources via the fecal-oral route; highly infectious and highly resistant to disinfection

d. Symptoms, if they occur, are produced by trophozoites after an incubation period of 1–2 weeks; some patients are asymptomatic.

e. Risk factors

(1) Immunocompromised patients (especially those with AIDS)

(2) Homosexual men

(3) Animal handlers

(4) Children attending daycare centers

f. Clinical presentation

(1) Profuse watery diarrhea, abdominal cramping, anorexia, nausea, and flatulence

(2) In immunocompetent patients, these symptoms may persist for 1–3 weeks and result in significant dehydration, but they are generally self-limited.

(3) In immunocompromised patients, malabsorption and significant weight loss occurs as well; symptoms may persist for months to years, producing significant discomfort and morbidity.

g. Diagnostic evaluation
   (1) Fecal studies for oocysts using an acid-fast stain or enzyme immunoassay testing *Cryptosporidium* (> 95% sensitive); must specify to the laboratory that the sample to be tested for *Cryptosporidium*
   (2) Fecal blood and leukocytes are usually absent.
   (3) Eosinophilia is absent.
h. Management
   (1) Immunocompetent patients
      (a) Rehydration and symptomatic therapy
      (b) Nitazoxanide may hasten recovery.
   (2) Immunodeficient patients are resistant to treatment.
      (a) When possible, therapy should be directed toward correcting the underlying immunodeficiency.
      (b) Nitazoxanide or paromomycin plus azithromycin may decrease symptoms in these patients, but is not curative.
      (c) In AIDS patients, initiation of highly active antiretroviral therapy (HAART) is probably the best treatment, because it directly increases host immunity by suppressing HIV replication.

4. ***Cystoisospora* (formerly *Isospora belli*)**
   a. Coccidian parasite
   b. Transmitted by cysts via the fecal-oral route
   c. Usually an opportunistic infection, it is seen most commonly in patients with AIDS (especially in developing countries); most common in tropical and subtropical regions.
   d. Clinical presentation
      (1) Symptoms, when present, begin after an incubation period of 1–2 weeks and last for 2–6 weeks; some immunocompetent patients are asymptomatic, whereas immunocompromised patients tend to have a severe protracted course.
      (2) The typical patient is a 30-year-old Haitian male with a history of AIDS who presents with profuse watery diarrhea, abdominal cramps, nausea and vomiting. He also complains of weight loss and has signs of malabsorption. Fecal examination reveals oocysts.
   e. Diagnostic evaluation
      (1) Diagnosis is usually made by identifying oocysts in the feces, performing acid-fast staining of a fecal specimen, or identifying intracellular forms on biopsy of the small bowel.
      (2) Leukocytosis with moderate eosinophilia occurs in 50% and is an important clue to diagnosis.
   f. Management
      (1) TMP-SMX is the drug of choice; prolonged therapy is required in AIDS patients.
      (2) Pyrimethamine, nitrofurantoin, or furazolidone may be used in patients allergic to sulfa.

5. *Cyclospora*
   a. Coccidial parasite found in tropical and subtropical regions.
   b. Affects both immunocompetent and immunocompromised individuals

c. North American outbreaks have been traced to foods grown in tropical environments.
d. Disease occurs after eating or drinking contaminated food or water. Person-to-person transmission is unlikely.
e. Symptoms include sudden onset of explosive watery diarrhea with abdominal cramping after an incubation period of 1–2 weeks. Severe diarrhea lasts 2–3 days then becomes intermittent. Fatigue and weight loss are constant features.
f. Disease is self-limited in healthy patients but may last 6–8 weeks, and up to a year in immunocompromised patients.
g. Diagnosis is made by examination of feces for the oocysts; can be easily confused with *Cryptosporidium*.
h. Management
(1) TMP-SMX is the drug of choice.
(2) Ciprofloxacin may be useful in allergic patients.

6. *Necator americanus* (hookworm)
a. Infection occurs when the hookworm larvae penetrate through intact skin on contact with feces-contaminated soil.
b. Most common in warmer climates (tropics, southeastern United States) where people walk around barefoot and sanitation is poor.
c. After penetrating the skin, the larvae enter the bloodstream, ascend the trachea, descend to the esophagus to differentiate into adult worms, and migrate to the upper intestine where they attach to the mucosal wall and feed on host blood.
d. Clinical presentation
(1) Intermittent diarrhea, epigastric discomfort, weakness, low-grade fever, cough, rash, and weight loss
(2) Hypochromic, microcytic anemia may develop in children who are heavily infected.
e. Diagnostic evaluation
(1) Identify ova in the feces.
(2) The presence of hypochromic, microcytic anemia and marked eosinophilia further support the diagnosis.
f. Management
(1) Mebendazole, albendazole, or pyrantel pamoate
(2) An iron supplement

7. *Enterobius vermicularis* (pinworm)
a. Transmission is via ingestion of *E vermicularis* eggs.
b. Once ingested, eggs develop into adult worms in the large bowel; then the gravid females migrate to the anus at night to deposit their eggs.
c. Outbreaks often occur in schools, daycare centers, and other institutional settings.
d. Clinical presentation
(1) Patients most commonly complain of pruritus ani, particularly at night.
(2) Secondary skin changes and bacterial infection may also be present.
(3) Other associated problems may include insomnia, enuresis, urinary tract infections, and vaginitis.

e. Diagnostic evaluation
   (1) Identify either adult worms migrating in the perineal area or eggs on a cellophane tape swab of the anus.
   (2) Eosinophilia is absent.
f. Management
   (1) A single dose of mebendazole or pyrantel pamoate that is repeated again after 2 weeks
   (2) All family members should be treated.

E. Diarrhea in the AIDS patient

1. Patients who are HIV-positive are more susceptible to the typical enteric organisms and to many opportunistic organisms as well. In addition, it is believed that HIV itself may produce an enteropathy.
   a. Cytomegalovirus and *Cryptosporidium* are the two most common causes of diarrhea in these patients. *Mycobacterium avium* is another important cause.
   b. Multiple organisms are responsible in some patients (up to 25%).
   c. HIV-positive patients on the HAART regimen living in developed countries are more likely to have diarrhea related to their medications (most commonly protease inhibitors) than to an infectious cause. When diarrhea is the sole complaint of a patient on the HAART regimen, an infectious cause is found in <20%.
2. Unlike diarrheal illness in immunocompetent patients, diarrheal illness in AIDS patients is not a self-limiting disease. Because of decreased immunity and poor baseline nutritional status of these patients, diarrheal illness can have devastating consequences that require a more aggressive approach in evaluation and treatment.
   a. Chronic, high-volume watery diarrhea is most often due to a coccidial species such as *Cryptosporidium* or *Cystoisospora*.
   b. Cytomegalovirus and *Mycobacterium avium* produce chronic illness in which fever, weight loss, and abdominal pain are prominent. Diarrhea is mild to moderate.
   c. Initial evaluation should include fecal culture for *Salmonella, Shigella, Yersinia, Campylobacter*, and STEC.
   d. Feces should be examined for *Giardia, Entamoeba, Cryptosporidium, Isospora, Cyclospora, Mycobacterium*, and *C difficile*.
   e. Proctosigmoidoscopy may be required for severe symptoms.
   f. Empiric antimicrobials should not be administered.

VIII. BILIARY TRACT DISORDERS

A. General information

1. Bile is produced in the canaliculi of biliary tract. While fasting, 50% of bile is stored in the gallbladder and 50% flows into the duodenum.
2. Food in the stomach results in release of cholecystokinin-pancreozymin and vagal impulses, which induce gallbladder contraction.
3. Symptoms related to biliary disease are a frequent reason for emergency department visits.

B. Cholelithiasis

1. Gallstones
   a. 20% of women and 8% of men have gallstones.
   b. The vast majority of gallstones are cholesterol stones.
   c. Risk factors for cholesterol stones
      (1) Female
      (2) Obesity
      (3) Increased parity
      (4) Advancing age
      (5) Rapid weight loss
      (6) Cystic fibrosis
      (7) Medications: oral contraceptives
      (8) Family tendency: very high rate in Pima Indians
   d. Pigmented stones
      (1) Black: associated with hemolysis such as sickle cell disease
      (2) Brown: associated with infection (bacterial as well as with *Ascaris, Clonorchis*)
2. Clinical features: biliary colic
   a. Pain often colicky in nature.
   b. Right upper quadrant pain (may be felt over epigastrium as well)
   c. Many patients perceive steady constant pain.
   d. Associated with nausea and vomiting
   e. May radiate to scapula or shoulder
3. Diagnostic evaluation
   a. ALT and AST to assess for hepatitis
   b. Bilirubin and alkaline phosphatase to assess for duct obstruction
   c. Lipase to determine if pancreatitis present
   d. Ultrasound is the test of choice.
4. Management
   a. Correction of fluid and electrolyte abnormalities if vomiting present
   b. Relief of nausea and vomiting
   c. Pain control
      (1) Anticholinergics to decrease spasm: glycopyrrolate
      (2) NSAIDs: ketorolac IV frequently useful
      (3) Opiates as needed
   d. Definitive treatment is cholecystectomy.

C. Cholecystitis

1. **Acute cholecystitis represents acute inflammation of the gallbladder.**
2. **Most cases result from acute obstruction of the cystic duct; 95% of patients will have gallstones identified. Duct obstruction may also be related to tumor, lymphadenopathy, fibrosis, parasitic disease.**
3. **Risk factors are identical to those of cholelithiasis.**

4. Clinical presentation
   a. Patients usually present with right upper quadrant pain that may be colicky but often becomes constant.
   b. Fever may be present.
   c. Nausea and vomiting are common.
5. Diagnostic evaluation
   a. Ultrasonography showing stones, thickened gallbladder wall, and pericholecystic fluid is virtually diagnostic.
   b. Absence of stones has a very high negative predictive value.

D. Variations and complications of cholecystitis
1. Acalculous cholecystitis (5% of cases)
   a. Occurs in postpartum/postoperative patients and those with burns or other major trauma, vascular disease, diabetes, sepsis, and CHF
   b. More common in the elderly
   c. Gangrene and perforation are relatively frequent in contrast to calculous cholecystitis.
2. Calculous cholecystitis is the most frequent cause of acute pancreatitis: a gallstone passes into the common bile duct and may occlude the pancreatic duct at the sphincter of Oddi.
3. Ascending cholangitis
   a. Occurs when stones passing through the common bile duct cause a purulent infection within the bile ducts that extends into the liver
   b. Clinical presentation
      (1) Fever and chills, abdominal pain, and jaundice (Charcot triad)
      (2) In severe cases, mental confusion and shock may also be present (Reynold pentad).
   c. This is a surgical emergency with a high mortality rate requires emergent decompression through open surgery, percutaneous drainage, or endoscopic retrograde cholangiopancreatography.
4. Emphysematous cholecystitis (rare)
   a. Infection with gas-forming bacteria (*E coli, Clostridium perfringens, Klebsiella,* and/or anaerobic streptococci)
5. Empyema of the gallbladder (an advanced stage of cholecystitis)
   a. Bacterial invasion of the gallbladder wall
   b. Leads to gross suppuration
6. Gallstone ileus (uncommon)
   a. Mechanism: a large gallstone erodes into the duodenum, lodges in the terminal ileum, and produces intestinal obstruction.
   b. Most commonly seen in elderly women (causes 25% or all small-bowel obstructions in this group) with multiple underlying medical problems and a history of gallstones.
   c. Diagnostic evaluation: radiographic findings show pneumobilia (air in the biliary tree), evidence of mechanical small-bowel obstruction, and a stone in the GI tract.

E. Clinical presentation

1. History
   a. Family history of gallbladder disease (common)
   b. Fatty food intolerance (common)
   c. Nausea (common)
   d. Bile-stained vomitus (variable because vomiting may not be present)
   e. Abdominal pain: most common presenting complaint, often precipitated by ingestion of a fatty or heavy meal. Initially, the pain is visceral in nature and felt in the midline. With prolonged obstruction of the cystic duct, somatic fibers become involved and the pain localizes to the right upper quadrant.
      (1) Distention of the gallbladder (due to obstruction of the cystic duct by a gallstone) → epigastric pain
      (2) Prolonged obstruction of the cystic duct → increased pressure within the gallbladder → chemical and/or bacterial inflammation of the gallbladder wall → right upper quadrant pain (which may radiate to the interscapular area, right scapula, or shoulder)
2. Physical examination
   a. Tachycardia and fever
   b. Tenderness in the right upper quadrant or epigastrium
   c. Murphy's sign: → pain and transient arrest of inspiration when direct pressure is applied to the right upper quadrant while the patient is taking a deep breath

F. Diagnostic evaluation

1. Laboratory abnormalities
   a. Mild leukocytosis with a left shift
   b. Bilirubin and alkaline phosphatase (may be normal)
      (1) Mildly increased → cholecystitis alone
      (2) Greatly increased → common duct stone
   c. Significantly increased serum amylase or lipase → pancreatitis due to a common duct stone
2. Radiographic abnormalities
   a. Plain films of the abdomen are usually negative or demonstrate nonspecific findings. Positive findings, when present, include:
      (1) Radiopaque gallstones (10%–15%)
      (2) Sentinel loop in the right upper quadrant
      (3) Air or an air–fluid level in the gallbladder → emphysematous cholecystitis
      (4) Air in the biliary tree → biliary enteric fistula
   b. Ultrasonography
      (1) Most useful initial test for imaging the gallbladder in the emergency setting
      (2) It is rapid, noninvasive, and can identify the following:
         (a) Thickness of the gallbladder wall (≥5 mm is diagnostic of cholecystitis)
         (b) Gallstones
         (c) Common duct stones
         (d) Dilated common duct

(e) Pericholecystic fluid

(f) Other sources of right upper quadrant pain (pathology of the liver, pancreas, and kidneys)

c. Nuclear scintigraphy

(1) Most accurate test for establishing diagnosis of cholecystitis

(2) Indicated if no stones are seen on ultrasound or there was equivocal thickness of the gallbladder wall

(3) If the isotope does not pass into the gallbladder within 1 hour of ingestion, the cystic duct is presumed to be obstructed.

d. CT scanning rarely offers any advantage over ultrasound, except that it may visualize common duct stones better.

G. Management

1. Patients with acute cholecystitis (fever, leukocytosis, significant abdominal tenderness, and vomiting) and/or its complications (pancreatitis, ascending cholangitis, jaundice) should be admitted.
2. Initial therapy should consist of:
   a. IV fluids
   b. Antiemetics and nasogastric tube suction (if persistent vomiting is present)
   c. Surgical consult
   d. Pain relief
   e. Broad-spectrum IV antibiotics
      (1) A second- or third-generation cephalosporin is adequate in most cases.
      (2) Triple antibiotic therapy (ampicillin, an aminoglycoside, and clindamycin or metronidazole) is indicated in the presence of sepsis.
3. Definitive therapy is cholecystectomy at the earliest convenient time (usually within 72 hours)

IX. HEPATITIS

A. Viral hepatitis

1. **Sequence of events: hepatic enzymes begin to increase before the prodromal phase → prodromal symptoms (usually constitutional) for 1–2 weeks→ icteric phase (when present) may be preceded by pruritus and dark urine followed by jaundice and possibly right upper quadrant pain with hepatomegaly or splenomegaly → recovery phase (symptoms resolve and hepatic enzymes normalize). In most cases, there is complete** recovery in 3–4 months. (Note that most patients do not develop jaundice.)
2. Hepatitis A
   a. **Caused by hepatitis A virus (HAV), an RNA virus spread by the fecal-oral route directly via person-to-person contact or indirectly through ingestion of contaminated water or food (particularly raw or undercooked shellfish)**
   b. **Illness is generally mild with a 15–50 day incubation period.**
   c. **Fecal shedding of the virus is maximal late in the incubation period and early in the prodromal phase.**

d. There is no carrier state, and it does not cause chronic liver disease.

e. Serologic marker is anti-HAV.

(1) Anti-HAV IgM indicates acute infection.

(2) Anti-HAV IgG indicates prior infection and confers immunity.

3. Hepatitis B

a. Caused by hepatitis B virus (HBV), a DNA virus spread primarily by the percutaneous route but may also be transmitted by intimate contact via blood, semen, or saliva

b. Illness is usually more severe and protracted than that produced by hepatitis A and has an incubation period of 45–160 days (mean 120 days); fulminant hepatic failure develops in 1% of cases (rapidly rising bilirubin levels, coagulopathy, and encephalopathy), and 80% of comatose patients die.

c. Chronic hepatitis or a chronic carrier state develops in 90% of infected neonates and in 10% of infected adults. These chronic carriers are all at increased risk of developing hepatocellular cancer; those with circulating virus continue to be a source of possible transmission.

d. Serologic markers in hepatitis B infection

(1) Hepatitis B surface antigen (HB_sAg) represents the outer protein coat of the virus. In most cases (>90%), it is detectable in the serum before hepatic enzymes increase and clinical symptoms appear; it lasts ~6 months. Its presence implies active hepatitis or the carrier state but not necessarily infectivity.

(2) Antibody to HB_sAg (anti-HB_s) appears in the serum 2–6 months after HB_sAg disappears; it is a sign of previous infection or vaccination and indicates immunity. Chronic carriers usually have persistent HB_sAg in their serum and, therefore, do not develop anti-HB_s.

(3) Hepatitis B core antigen (HB_cAg) (the core of the virus) is present only in hepatocytes, not in serum. Its presence, in conjunction with a negative anti-HB_s is suggestive of early infection.

(4) Antibody to HB_cAg (anti-HB_c) is present in the serum ~2 weeks after HB_sAg appears. It may be the only marker of recent infection during the "window period" between the disappearance of HB_sAg and the appearance of anti-HB_s. The presence of IgM anti-HB_c in high titer indicates acute infection and high infectivity, whereas the presence of IgG anti-HB_c, in association with anti-HB_s, implies remote infection.

(5) Hepatitis B epsilon antigen (HB_eAg) is found in serum containing Hb_sAg. Its presence indicates ongoing viral replication and high infectivity. The antigen disappears in those patients who recover from hepatitis B but persists in those who develop chronic hepatitis.

(6) Antibody to HB_eAg (anti-HB_e) appears during the acute phase of hepatitis B and implies that the patient is not likely to be infective. Anti-HB_e persists several months after HB_eAg disappears.

(7) Sound confusing? Well, it is, especially because the presence of active infection does not necessarily correlate with infectivity, or how likely the patient is to transmit the disease.

Table 8: One Method of Evaluating Serologic Results

No infectivity	Low infectivity	Infectivity	High infectivity
+ anti-HB_s	+ anti-HB_e	+ HB_sAg	+ HB_eAg

(8) Only two serologic markers (HB_sAg and IgM anti-HB_c) are actually needed to establish the diagnosis of infection with hepatitis B and determine its chronicity; HB_sAg confirms that infection is present, while IgM anti-HB_c implies that it is acute.

e. The CDC believes that successful hepatitis B vaccination (ie, that which is documented by anti-hepatitis B surface antibodies ≥10 IU at any time during a patient's life) confers lifetime protection. Therefore, do not test the source of (or recipient of) a blood exposure (eg, needle stick) if the patient has been successfully immunized. Otherwise, perform routine testing (including antihepatitis B surface antibodies).

4. Hepatitis C
   a. Caused by hepatitis C virus (HBC) and seems to spread in a fashion similar to that of hepatitis B
   b. Most common cause of viral hepatitis in the United States
      (1) Prior to 1992, most cases occurred in association with the transfusion of blood/blood products; thus, hepatitis C was referred to as "post-transfusion hepatitis." Since the introduction of a marker in 1992 to screen blood for hepatitis C (anti-HCV), the number of transfusion-related cases of hepatitis C has been declining.
      (2) Currently, IV and intranasal drug abuse accounts for a growing number of new infections with hepatitis C.
   c. Incubation period is 15–160 days (mean = 50 days); the illness is similar to (but milder than) that caused by hepatitis B.
   d. Chronic hepatitis develops in 50%–85% of these patients, 30% of whom go on to develop cirrhosis. The delayed development of hepatocellular cancer in these cirrhotic patients is not uncommon.
   e. Anti-HCV is the serologic marker for hepatitis C; it is detectable in the serum 1–6 months after the onset of symptoms; anti-HCV indicates the presence of chronic infection and potential infectivity.
5. Hepatitis E
   a. Caused by hepatitis E virus, an RNA virus; incubation period is 15–60 days.
   b. Resembles hepatitis A in its mode of transmission and the clinical course but is associated with a higher incidence of fulminant liver failure and mortality, particularly in pregnant patients.
   c. Chronic infection does not occur.
   d. A serologic marker has not yet been identified.
6. Hepatitis delta virus (HDV)
   a. A "defective" RNA-containing virus that can only occur in patients who are currently infected with HBV. Infection may occur concomitantly with an acute HBV infection or as a superinfection in patients with chronic hepatitis B.
   b. Patients with acute concomitant infection have a better prognosis than those with superinfection; the illness is generally self-limited.
   c. Superinfection is associated with a high mortality rate (relative to acute HBV infection) and frequent development of chronic HDV hepatitis with subsequent cirrhosis.
   d. The serologic marker is anti-HDV.

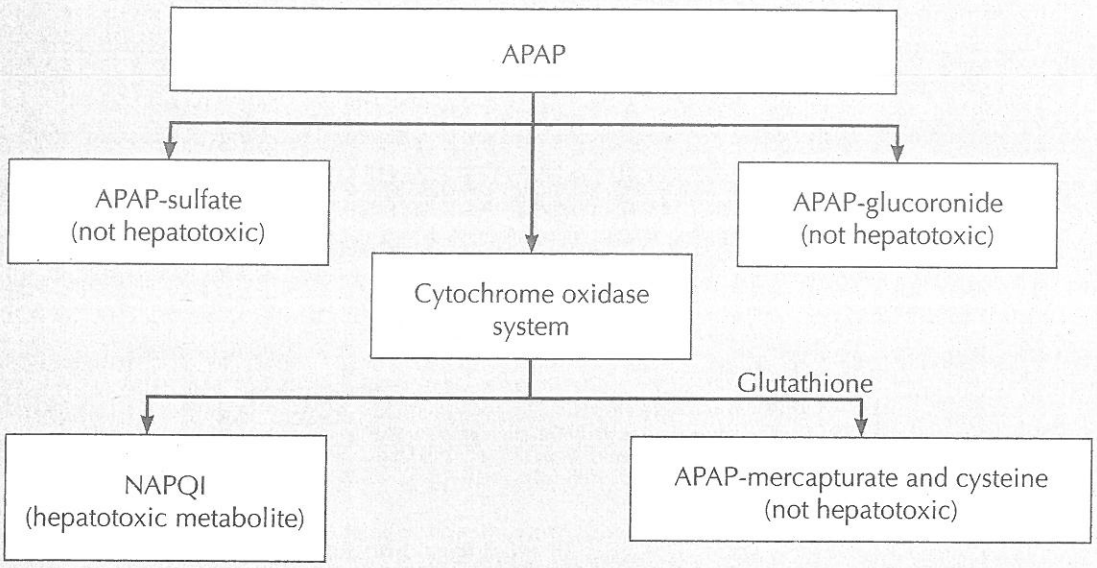
7. Diagnostic evaluation
   a. Serum aminotransferases are increased; AST is 10–100 times normal and ALT is usually higher than AST.
   b. Bilirubin (total and direct) is increased.
   c. Prothrombin time is usually normal; prolongation points to a more complicated course and a poorer prognosis.
8. Prevention and prophylaxis
   a. Immune globulin, formerly known as gamma globulin or serum immune globulin, is 80%–90% effective in preventing infection with HAV if administered within 14 days of exposure.
   b. Postexposure prophylaxis with immunoglobulin is required for close personal contacts (home, daycare, etc). Exposed school, hospital, workplace contacts do not require prophylaxis.
   c. Hepatitis A vaccine should be recommended for those traveling to endemic areas. The CDC recommends the vaccine be administered at least 2 weeks before travel to endemic areas. Immunity develops by 20 days after administration. Travelers who cannot receive the vaccine should receive immune globulin before travel for passive immunity that will last 3 months.
   d. Hepatitis B immune globulin (HBIG) and hepatitis B vaccine have both been demonstrated to prevent hepatitis B; HBIG confers passive immunity, while the vaccine confers active immunity. For maximal effectiveness, HBIG must be administered within 7 days of exposure; the sooner the better. High-risk exposure (percutaneous/mucosal from a known HB_sAG+ individual) in patients who have completed the HBV vaccine series who are known to be HB_sAB+ do not require HBIG. If unknown response, can test for presence of surface antibody. If negative, should receive HBIG. Nonresponders to the vaccine should be treated as unimmunized and receive HBIG.
   e No vaccine is available for hepatitis C. Postexposure prophylaxis with immune globulin is not recommended, and results with interferon are inconclusive.
      (1) For potential exposures, the CDC recommends determining the serologic status of the source case; however, the average incidence of anti-HCV seroconversion after unintentional needle stick or other sharp exposure from an HCV-positive source is only 1.8% (range 0–7%)
      (2) The exposed worker should have baseline hepatitis C antibody tests and liver function tests (including AST and ALT), which should be repeated in 6 months.
   f. There is no specific vaccine for hepatitis delta. The best preventive measure is vaccination for hepatitis B, because infection with hepatitis delta cannot occur in the absence of infection with hepatitis B.
   g. A hepatitis E vaccine has been very promising in trials but is not yet commercially available.
9. Indications for admission
   a. Refractory vomiting
   b. Fluid or electrolyte imbalance
   c. Prolonged prothrombin time (>3–5 seconds compared with control)
   d. Bilirubin >20–30 mg/dL
   e. Hypoglycemia
   f. Immunosuppression

g. Severe underlying disease
h. Early signs of encephalopathy (eg, agitation, altered mental status)
i. Age >45–50 years old

B. Toxic hepatitis

1. Hepatotoxins that can produce morphologic changes in the liver and resemble those of acute viral hepatitis
   a. Halothane
      (1) Produces toxicity via a toxic metabolite and a hypersensitivity reaction
      (2) Most cases (75%) occur in patients who have been exposed to halothane in the past.
      (3) More common in adults (particularly women) and obese individuals.
      (4) Postoperative fever, rash, and eosinophilia may be present.
      (5) Onset is abrupt; usually occurs a couple of days after exposure.
      (6) Mortality rate is 20%–40% in severe icteric cases.
   b. Methyldopa
      (1) Produces injury via a toxic metabolite and a hypersensitivity reaction.
      (2) Mild transient increase of transaminase levels is seen in ~5% of patients treated with this drug, and <1% actually develop acute hepatitis.
      (3) Development of a rash, arthralgias, and lymphadenopathy may precede onset of jaundice.
      (4) Clinical improvement occurs when methyldopa is discontinued, but cases of chronic hepatitis and cirrhosis have been described.
   c. Isoniazid (INH)
      (1) Produces toxicity via a toxic metabolite
      (2) Incidence of developing toxic hepatitis is age-related; it is rare in patients <20 years old but approaches 3% in those >50 years old.
      (3) Susceptibility is potentiated by alcoholism and by concurrent use of rifampin or pyrazinamide.
   d. Phenytoin
2. Hepatotoxins that produce cholestatic changes in the liver with clinical manifestations of malaise, anorexia, nausea, and vomiting
   a. Anabolic steroids
   b. Oral contraceptives
   c. Chlorpropamide
   d. Chlorpromazine
   e. Erythromycin estolate
   f. Haloperidol
   g. Verapamil
   h. Phenobarbital
3. Hepatotoxins that can produce massive hepatic necrosis

a. **Acetaminophen (APAP)**



Mechanism of Toxicity of Acetaminophen

↑ dose of APAP → saturation of the sulfate and glucuronide pathways and depletion of glutathione → buildup of toxic metabolite (NAPQI)

b. Carbon tetrachloride
c. Yellow phosphorus
d. Mushrooms (eg, Amanita phalloides)

X. ALCOHOLIC LIVER DISEASE

A. Syndromes

1. Hepatic steatosis (fatty liver)
   a. Consumption of even moderate amounts of alcohol can lead to deposition of fat within hepatocytes.
   b. Clinical presentation: nontender hepatomegaly associated with mild increase in serum transaminase
   c. Resolves in 4–6 weeks with abstinence from alcohol
2. Alcoholic hepatitis
   a. Clinical presentation
      (1) Ranges from a mild illness to acute liver failure, depending on the degree of hepatocellular necrosis and intrahepatic inflammation
      (2) Presence of encephalopathy is the strongest predictor of short-term mortality. Other scoring systems are based on presence of jaundice and coagulopathy.

(3) Patients classically present with generalized weakness, anorexia, nausea, abdominal discomfort, and weight loss.

(4) Urine may be dark in color.

(5) Examination usually reveals a low-grade temperature, jaundice, and a tender, enlarged liver.

b. Diagnostic evaluation

(1) CBC shows macrocytic anemia, leukocytosis, and thrombocytopenia.

(2) Bilirubin and alkaline phosphatase levels are increased.

(3) AST and ALT levels are 2–10 times normal, with the AST level higher than the ALT level.

(4) INR >1.5 portends a complicated course.

3. Alcoholic (Laennec) cirrhosis

a. An irreversible disease that produces permanent histologic changes in the liver (hepatocytic nodules and fibrous tissue) that disrupt normal hepatic blood flow and result in portosystemic shunting with concomitant portal hypertension

b. Clinical presentation

(1) Patients typically present with chronic fatigue and anorexia; however, some remain relatively asymptomatic until they develop a complication (eg, hepatic encephalopathy, GI bleed).

(2) Classic clinical findings include jaundice, spider angiomata, palmar erythema, gynecomastia, muscle wasting, Dupuytren contracture, ascites, and pedal edema.

c. Diagnostic evaluation

(1) CBC: anemia, leukopenia, thrombocytopenia

(2) AST and ALT levels minimally increased

(3) Prolonged prothrombin time (useful predictor of severity of liver disease), INR >1.5

(4) Increased bilirubin (useful predictor of severity of liver disease) and alkaline phosphatase levels

(5) Hypoalbuminemia

(6) Hyponatremia

(7) Hypokalemia

B. Complications

1. Bleeding esophageal varices

a. 25%–40% of patients with cirrhosis bleed from varices, and each episode of hemorrhage carries a 30% mortality risk.

b. Clinical presentation: hemorrhagic shock with massive hematemesis

c. Diagnostic evaluation: emergency endoscopy should be performed as soon as possible to confirm the diagnosis, exclude other sources of bleeding, and provide therapy. (See also Esophageal Disorders, pages 227–234)

2. Portosystemic encephalopathy

a. Due to accumulation of toxic substances in the blood

b. Develops in patients with cirrhosis with extensive portosystemic shunting when the diseased liver is no longer able to perform its metabolic function of detoxifying wastes adequately

c. Precipitating factors
   (1) Azotemia (GI bleeding, high-protein diet)
   (2) Injudicious use of medications (sedatives, analgesics, and tranquilizers)
   (3) Hypokalemic metabolic alkalosis
   (4) Infection
   (5) Hypoxemia
   (6) Hypoglycemia
   (7) Dehydration

d. Clinical presentation
   (1) Altered level of consciousness, fetor hepaticus, and asterixis ("liver flap"—best demonstrated with the dorsiflexed wrist), along with other signs and symptoms of chronic liver disease
   (2) Serum ammonia level is usually increased, although levels correlate poorly with mental status.

e. Management
   (1) General supportive measures
   (2) Correction/elimination of precipitating factors
   (3) Administration of lactulose and/or neomycin (to eliminate ammonia)
   (4) Protein-restricted diet

3. Hepatorenal syndrome
   a. Syndrome of acquired renal failure of unknown cause that occurs in decompensated cirrhotic patients
   b. Pathogenesis: vasoconstriction and shunting of blood away from renal cortex → ↓ glomerular filtration rate → ↓ renal output → azotemia
   c. Prognosis is dismal, with a mortality rate of nearly 100%.

4. Spontaneous bacterial peritonitis
   a. Occurs in up to 33% of patients with liver disease and ascites
   b. Clinical presentation
      (1) Patients with liver disease who present with fever, abdominal tenderness, worsening ascites, and decreasing hepatic function.
      (2) Many have no symptoms; consider paracentesis for any patient with ascites ill enough to require hospital admission.
   c. Diagnostic evaluation
      (1) Evaluation of ascitic fluid (obtained via paracentesis) confirms the diagnosis.
      (2) A granulocyte count >500/mm^3 is very suggestive, with an absolute PMN count >250 cells/mm^3 diagnostic.
      (3) A positive culture is diagnostic.
   d. *E coli* and *Streptococcus spp* are the most common bacterial isolates.
   e. Treatment is with IV antibiotics (eg, a third-generation cephalosporin).
   f. Mortality rate ranges from 30% to 100%.

XI. PANCREATITIS

A. Etiology

1. Acute pancreatitis is most often due to ethanol ingestion or biliary tract disease (gallstones). Other causes include abdominal trauma, surgery, endoscopic retrograde cholangiopancreatography, drugs (thiazides, furosemide, azathioprine, tetracycline, sulfonamides), penetrating peptic ulcer, hyperlipidemia, hypercalcemia, infections (mumps, viral hepatitis, infectious mononucleosis, *Mycoplasma*), and pregnancy.
2. Chronic pancreatitis is the result of repeated episodes of acute pancreatitis and is usually due to chronic alcoholism of long duration (75% of cases in United States). Other causes include severe protein–calorie malnutrition, hyperthyroidism, and pancreas divisum.

B. Clinical presentation

1. Patients classically present after ingestion of a fatty meal or after binge drinking and complain of epigastric pain, nausea, and vomiting.
2. The epigastric pain is constant in nature with radiation directly into the back, and it is often eased when the patient leans forward.
3. Examination may reveal epigastric tenderness with guarding, tachycardia (due to fever, hypovolemia, and pain), tachypnea (due to pulmonary involvement), a low-grade fever, and hypoactive bowel sounds. However, because of the retroperitoneal location of the pancreas, rebound is generally absent.
4. Grey Turner sign (bluish discoloration of the flanks) and Cullen sign (bluish discoloration around the umbilicus) indicate the presence of retroperitoneal hemorrhage and point to the diagnosis of hemorrhagic pancreatitis; unfortunately, these signs are not usually present. They are also not specific for pancreatitis.

C. Diagnostic evaluation

1. Serum amylase and lipase levels are typically increased in acute pancreatitis; however, the degree of increase does not correlate with the severity of disease.
2. Very high levels are usually seen when biliary tract disease (gallstones) is the underlying cause.
3. However, pancreatic enzymes are not usually increased in patients with chronic pancreatitis, because their pancreas is "burned out."
4. Serum amylase
   a. Increases shortly after the onset of symptoms and returns to baseline levels within 1–4 days
   b. An increase 1.5 times the upper limit of normal typically indicates acute pancreatitis.
5. Serum lipase
   a. Found primarily in the pancreas (unlike serum amylase, which is released by several organs)
   b. As sensitive as serum amylase but has the advantage of being more specific; specificity is almost 100%.
   c. Assays are readily available, reliable, and inexpensive.
6. Other laboratory studies
   a. Glucose to exclude hyperglycemia
   b. Calcium to exclude hypocalcemia (most common laboratory abnormality)

c. WBC count: leukocytosis is common
d. Hematocrit (↓ hematocrit is suggestive of hemorrhagic pancreatitis)
e. BUN and creatinine usually increased due to hypovolemia.
f. AST and lactate dehydrogenase: significant increase suggests severe disease.

7. Plain radiographs are most helpful in excluding other diagnoses. Radiographic findings associated with pancreatitis include:
   a. Abdominal films
      (1) Scattered calcifications in the area of the pancreas are suggestive of chronic pancreatitis.
      (2) Evidence of ileus and air-trapping in the small bowel adjacent to the pancreas → sentinel loop (suggests acute pancreatitis)
      (3) Distended colon and collapse of the distal colon → colon-cutoff sign
   b. Chest radiograph
      (1) Left pleural effusion or elevated left hemidiaphragm
      (2) Findings consistent with ARDS may be seen in patients with severe disease.
   c. Ultrasound
      (1) Should be performed early if gallstone pancreatitis suspected
      (2) May miss common duct stones or dilatation of duct
      (3) Endoscopic ultrasound may be required.
   d. CT scan: used to identify other causes of abdominal pain, establish severity of disease, and exclude complications such as pancreatic necrosis, hemorrhage, vascular abnormalities, and pseudocyst or abscess
   e. MRI cholangiopancreatography: noninvasive and may help establish cause

D. Management: primarily supportive

1. Hydration
   a. Fluid resuscitation with normal saline as required. Most patients are dehydrated because of vomiting and fluid sequestration.
   b. Close monitoring of vital signs, urine output
   c. Electrolyte replacement as necessary
2. Pain control
   a. Morphine, hydromorphone are appropriate (despite potential to induce spasm of sphincter of Oddi, no adverse outcome in people has been demonstrated).
   b. Meperidine is the traditional choice but has no proven advantage.
3. Control of vomiting
   a. IV antiemetics as required
   b. Nasogastric suctioning only for patients with intractable vomiting or ileus
   c. No evidence to support fasting in mild to moderate pancreatitis
4. Nutritional support
   a. Enteral feeding if tolerated
   b. If enteral feeding not tolerated, consider parenteral feeding.
5. Monitoring for complications of acute pancreatitis

E. Poor prognostic signs (Ranson criteria)

1. On admission
   a. Age >55 years old
   b. Leukocytosis (WBC count >16,000/mm^3)
   c. Hyperglycemia >200 mg/dL
   d. AST > 250 U/L
   e. Lactate dehydrogenase >350 IU/L
2. 48 hours later
   a. Calcium <8 mg/dL
   b. PO_2 <60 mmHg (suggests ARDS)
   c. Fall in hematocrit >10% (suggests hemorrhagic pancreatitis)
   d Increase in BUN of >5 mg/dL
   e. Base deficit >4 mEq/L
   f. Sequestration >4 L of fluid
3. Mortality correlates directly with the number of poor prognostic signs present; the greater the number, the greater the mortality.

F. Complications

1. Pleural effusions (usually left-sided)
2. ARDS is due to deactivation of surfactant (most common cause of death due to acute pancreatitis)
3. Pancreatic phlegmon → pseudocyst
4. Pancreatic abscess → sepsis
5. Hemorrhage (intrapancreatic or intraperitoneal) → shock
6. Pancreatic ascites
7. Third spacing of fluids → hypovolemia → acute tubular necrosis
8. Disseminated intravascular coagulation

ABDOMINAL AND GASTROINTESTINAL DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A patient presents complaining of vomiting blood (may or may not be actively vomiting at time of presentation) and has dried blood on the lips. Heart rate is 175 beats per minute, and blood pressure is 60/30 mmHg.

What is the diagnosis?

Scenario B

Presentation: A patient presents with a history of ingesting a foreign body and complains of a sensation of something stuck in the throat. Symptoms include vomiting, gagging, choking, and dysphagia.

What is the diagnosis?

Scenario C

Presentation: A patient has had a gradual onset of periumbilical discomfort that subsequently localized to the right lower quadrant. Other symptoms include anorexia, nausea, and vomiting. The pain started before the vomiting.

Physical examination: Findings reveal right lower quadrant tenderness to palpation with positive McBurney and Rovsing signs.

What is the diagnosis?

Scenario D

Presentation: An elderly patient with new onset atrial fibrillation (controlled or uncontrolled) presents with sudden onset of abdominal pain. The patient is not anticoagulated.

Physical examination: Abdominal examination findings are generally nonspecific despite significant pain.

What is the diagnosis?

Scenario E

Presentation: A patient with known colonic diverticuli presents complaining of lower quadrant abdominal pain (left sided). The pain is crampy, vague, and poorly localized.

Physical examination: The physical examination is relatively benign. Pain localizes on palpation to the left lower quadrant or suprapubic region.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: upper GI bleeding

Management: Initial efforts are directed at determining hemodynamic stability and determining need for immediate resuscitation.

Unstable patients require two large-bore IV lines with fluid boluses of normal saline. Administer blood product transfusion as necessary. If patient is anticoagulated, may need immediate reversal as appropriate. Immediate endoscopy may be needed for control of esophageal or ulcer bleeding. Administer IV proton-pump inhibitor. Regardless of history, ulcer bleeding is more common than variceal bleeding, but if variceal bleeding is suspected, administer octreotide.

For stable patients, determine the source of bleeding. If actively vomiting, the source is obvious. If not vomiting, presence of melena, or nasogastric aspiration with presence of blood or "coffee grounds," is highly predictive of upper GI source. However, absence of blood via nasogastric tube does not exclude upper GI source. Bright red blood or blood clots per rectum are more predictive of lower GI source. Determine need for immediate intervention or admission. Blatchford score helps predict need for intervention.

Scenario B

Diagnosis: esophageal foreign body

Key facts: Most cases (80%) of ingested esophageal foreign bodies are in children. They may not be old enough to give history. The cricopharyngeus muscle (C6) is the most common site of obstruction in children <4 years old. Most food bolus impactions are in adults and are often associated with a history of gastroesophageal reflux disease (GERD), webs, strictures, etc. The lower esophageal sphincter (T10-11) is the most common site in adults.

Diagnostic evaluation: Patients with obstruction are usually drooling secretions (not swallowing) and tend to regurgitate swallowed liquids. Plain radiographs of neck and chest show radiodense foreign bodies. Radiolucent objects such as food, plastic, fish bones, toothpicks, etc, are unlikely to be seen; CT with water-soluble contrast may be needed for identification (more likely if object thought to be in cervical region). Esophagrams are no longer commonly done.

Management: Endoscopy is the treatment of choice for removal of food boluses and foreign bodies that have become lodged in the esophagus. IV glucagon may be used to relax smooth muscle at the lower esophageal sphincter and allow the food bolus to pass. Sharp objects should be removed before they pass the pylorus. *Button batteries must be removed immediately!*

Scenario C

Diagnosis: appendicitis

Key facts: Appendicitis is the most common cause for emergency surgery in the United States. Atypical presentations are much more common in young and elderly patients. Scoring systems have been developed that may assist in assessing likelihood o diagnosis. Physical examination may be atypical in many patients, depending on the location of the appendix.

Diagnostic evaluation: Laboratory studies may not be helpful. Strong consideration should be given to compression-graded ultrasonography in children as the initial imaging choice; if negative and the diagnosis is highly likely, follow with CT. Adults generally require CT of abdomen and pelvis. Some controversy exists over the use of oral contrast. Ultrasound is less useful in adults but may be helpful in women to exclude pelvic pathology.

Management: Immediate surgical consult is suggested, although current data support delayed surgery in stable patients. Antibiotic coverage is appropriate and should cover typical abdominal pathogens. Choices include cefoxitin, cefotetan, and piperacillin/tazobactam.

Scenario D

Diagnosis: mesenteric ischemia, embolic occlusion of the superior mesenteric artery from a thrombus in the atria

Diagnostic evaluation: Laboratory investigation is frequently not helpful early on. Serum lactate is usually increased but not until bowel infarction has occurred. Acidosis may be present late. Helical CT angiography of the abdomen is currently the test of choice, although some centers may still perform conventional angiography.

Alternative presentation: Another typical presentation is an elderly patient with a history of coronary artery disease who presents with diffuse abdominal pain. The patient may report having had abdominal pain after eating on prior occasions. This is "intestinal ischemia" due to vascular disease. Examination early on shows few specific findings. Pain is out of proportion to findings. Feces may be positive for guaiac; there may be gross blood per rectum.

Management: Fluid resuscitation as required. If pressors are needed, use positive inotropes rather than vasoconstrictors. If nonocclusive interventional radiology is done, may use papaverine injection; if occlusive, revascularization is required.

Scenario E

Diagnosis: diverticular disease

Key facts: Signs of systemic toxicity are absent unless diverticuli have perforated or abscess formation has begun. Peritoneal signs are unlikely unless the patient has complicated diverticular disease, which involves microperforation of the bowel wall with formation of phlegmon in the immediate area. Minority of these will have frank free air in abdomen.

Alternative presentation: Another less likely presentation consists of massive lower GI hemorrhage that is painless. This is responsible for up to 40% of lower GI hemorrhage in elderly patients.

Diagnostic evaluation: CBC, hematocrit, and blood type and screen should be done in patients with diverticular hemorrhage. WBC count is likely to be increased, particularly in complicated disease. If complicated diverticulitis is suspected, CT of the abdomen and pelvis should be done. (Neither colonoscopy nor barium enema are done in the acute setting.) Patients in whom diverticular disease is suspected but have no known history may also benefit from CT to establish the diagnosis as well as exclude other pathology. Patients with history of diverticulosis and mild symptoms may not require imaging in the emergency department.

Management: Patients with uncomplicated diverticulitis can be treated on an outpatient basis with pain control and antibiotic therapy. Acceptable regimens include ciprofloxacin + metronidazole, TMP-SMX + metronidazole, or amoxicillin-clavulanate for 10 days.

Patients with complicated diverticulitis need resuscitation as necessary, IV antibiotic therapy similar to the above or ticarcillin-clavulanate or ampicillin-sulbactam. Percutaneous drainage of the abscess may be required. A surgical consult should be obtained for all with free air.

NOTES

THORACIC AND RESPIRATORY DISORDERS

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THORACIC AND RESPIRATORY DISORDERS: SELF-ASSESSMENT QUESTIONS

1. The most common cause of community-acquired bacterial pneumonia is:

   (a) Group A ß-hemolytic streptococci
   (b) *Haemophilus influenzae*
   (c) *Streptococcus pneumoniae*
   (d) *Klebsiella pneumoniae*

2. A sputum Gram stain revealing encapsulated gram-positive lancet-shaped diplococci is most consistent with:

   (a) *Staphylococcus aureus*
   (b) *Haemophilus influenzae*
   (c) *Mycoplasma pneumoniae*
   (d) *Streptococcus pneumoniae*

3. Although abscess formation is not common, it can be seen in association with each of the following causes of pneumonia except:

   (a) *Klebsiella pneumoniae*
   (b) *Chlamydia pneumoniae*
   (c) *Pseudomonas aeruginosa*
   (d) *Staphylococcus aureus*

4. The antiviral agent used to treat infants and children with severe respiratory syncytial virus pneumonia or bronchiolitis is:

   (a) Rimantadine
   (b) Ribavirin
   (c) Adenosine arabinoside
   (d) Amantadine

5. A 55-year-old male smoker presents in August with signs of an atypical pneumonia, relative bradycardia, and GI complaints. He is on no medications. The most probable cause of the pneumonia is:

   (a) *Mycoplasma pneumoniae*
   (b) *Francisella tularensis*
   (c) *Legionella pneumophila*
   (d) *Coxiella burnetii*

6. All of the following statements regarding *Klebsiella* pneumonia are true except:

   (a) Sputum Gram stain reveals encapsulated gram-positive organisms that are seen in pairs.
   (b) The sputum is thick and brown in color, resembling currant jelly.
   (c) Patients with alcoholism, diabetes, or COPD are most commonly affected.
   (d) Chest radiograph commonly reveals a necrotizing right upper lobe infiltrate.

7. Which of the following statements regarding the TB skin test is most accurate?

   (a) It is positive in 100% of patients with TB meningitis.
   (b) A positive reaction indicates the presence of infection with *Mycobacterium tuberculosis* but not necessarily the presence of active disease.
   (c) It is always positive in patients infected with *M tuberculosis*.
   (d) Criteria for interpreting the test as positive are the same for all patients regardless of background/concurrent illnesses.

8. Isoniazid therapy for TB is associated with the multiple complications listed below. Which one of these complications can be prevented or minimized by the simultaneous administration of pyridoxine?

   (a) Phenytoin toxicity
   (b) Hepatitis
   (c) Hypersensitivity reaction
   (d) Peripheral neuritis

9. On which of the following views of the chest is a small pleural effusion most likely to be detected?

   (a) Supine
   (b) PA
   (c) Lateral decubitus
   (d) Lateral

10. All of the following are appropriate in the acute treatment of aspiration pneumonia except:

    (a) Administration of steroids and prophylactic antibiotics
    (b) Administration of supplemental oxygen
    (c) Bronchoscopy to remove large particles
    (d) Use of continuous positive-airway pressure (CPAP)

11. The severity of pulmonary injury resulting from aspiration of foreign material is determined by all of the following factors except:

    (a) The presence of bacterial contamination
    (b) The pH and volume of the aspirate
    (c) The presence of particulate matter
    (d) The position of the patient at the time of aspiration

12. A pneumothorax may be detected on ultrasound when which of the following is seen?

    (a) B lines
    (b) Seashore sign in M-mode
    (c) Lung rockets
    (d) Loss of pleural sliding

13. Signs and symptoms of a tension pneumothorax may include all of the following except:

    (a) Jugular venous distention, cyanosis, and dyspnea
    (b) Hypertension
    (c) Hyperresonance to percussion and absence of breath sounds on the affected side
    (d) Deviation of the trachea to the contralateral side

14. Which modality assesses the degree of airflow obstruction in the asthmatic patient?

    (a) Arterial blood gases
    (b) Pulmonary function tests (PEFR or FEV_1)
    (c) Pulse oximetry
    (d) Chest radiograph

15. The initial treatment of choice for the asthmatic patient is:

    (a) Corticosteroids
    (b) Atropine
    (c) Inhaled ß-adrenergic agents
    (d) Subcutaneous ß-adrenergic agents

16. All of the following are accurate indicators of a severe asthmatic attack except:

    (a) The presence of wheezing
    (b) The use of accessory muscles
    (c) The presence of diaphoresis and cyanosis
    (d) The presence of a pulsus paradoxus >12 mmHg

17. All of the following statements regarding the use of ß-adrenergic agonists in the treatment of asthma are accurate except:

    (a) Their primary effect is on the large central airways.
    (b) They promote bronchodilation by increasing cyclic AMP.
    (c) Their onset of action is <5 minutes.
    (d) Agents with $ß_2$-selectivity are preferred.

18. All of the following are causes of noncardiogenic pulmonary edema except:

(a) Fat embolus
(b) Fluid overload
(c) Drug overdose
(d) Multiple trauma

19. All of the following statements regarding noncardiogenic pulmonary edema are true except:

(a) The heart size is small or normal.
(b) Pulmonary arterial wedge pressure is increased.
(c) Chest radiograph shows bilateral pulmonary infiltrates.
(d) Lung compliance is reduced.

20. Pneumonia is most commonly acquired via _____.

(a) Hematogenous spread from another site
(b) Direct introduction of organisms into the pleura or lungs
(c) Aspiration of oropharyngeal secretions
(d) None of the above

21. All of the following statements regarding pneumococcal pneumonia are accurate except:

(a) Patients usually give a history of a single shaking chill followed by the development of a cough productive of rust-colored sputum.
(b) The WBC count is usually $<12{,}000/mm^3$.
(c) A pleural effusion may be present in up to 25% of patients.
(d) Penicillin is the treatment of choice.

22. The serotype of *Haemophilus influenzae* that is responsible for 95% of human infections is:

(a) Type A
(b) Type B
(c) Type C
(d) Type D

23. Atypical pneumonias are characterized by all of the following except:

(a) Abrupt onset
(b) Moderate fever
(c) Presence of constitutional symptoms
(d) Nonproductive cough

24. A pet shop employee presents with severe headache, malaise, myalgias, and cough. Examination reveals a fever of 104°F (40°C), hepatosplenomegaly, and a relative bradycardia. Laboratory evaluation reveals patchy perihilar infiltrates on chest radiograph, increased liver function tests, and proteinuria. The organism most likely responsible for this patient's pneumonia is:

    (a) *Coxiella burnetii*
    (b) *Francisella tularensis*
    (c) Hantavirus
    (d) *Chlamydia psittaci*

25. The agent of choice for the treatment of the patient described in the question above is:

    (a) Tetracycline
    (b) Penicillin
    (c) Chloramphenicol
    (d) Ribavirin

26. ARDS most commonly occurs in association with:

    (a) Aspiration
    (b) Trauma
    (c) Sepsis
    (d) Massive blood transfusions

27. Central cyanosis may be caused by all of the following conditions except:

    (a) High altitude
    (b) Methemoglobinemia
    (c) CHF
    (d) Anatomic shunts

ANSWERS

1. c	7. b	13. b	19. b	25. a
2. d	8. d	14. b	20. c	26. c
3. b	9. c	15. c	21. b	27. c
4. b	10. a	16. a	22. b	
5. c	11. d	17. a	23. a	
6. a	12. d	18. b	24. d	

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. PNEUMONIA

A. Bacterial pneumonia (most common cause of a focal infiltrate)

1. Epidemiology
   a. Accounts for up to 10% of hospital admissions in the United States
   b. Most pneumonias are the result of a single species of bacteria.
      (1) *Streptococcus pneumoniae*
      (2) *Haemophilus influenzae*
      (3) *Klebsiella pneumoniae*
      (4) *Staphylococcus aureus*
      (5) *Escherichia coli*
      (6) *Pseudomonas aeruginosa*
      (7) Group A streptococci
      (8) *Moraxella catarrhalis*
   c. Mechanisms of infection
      (1) Aspiration of oropharyngeal secretions is the primary mechanism of acquisition.
         (a) 50%–70% of normal healthy individuals aspirate in their sleep.
         (b) An abnormal swallowing mechanism (eg, from an altered mental status) or GI disease can lead to increased frequency of aspiration.
      (2) Other mechanisms
         (a) Hematogenous spread from another site
         (b) Colonization
         (c) Direct introduction of organisms into the pleura or lungs, both of which are uncommon
   d. Predisposing factors
      (1) Impaired cough and gag reflex
         (a) Altered mental status
         (b) Seizures
         (c) Syncope
         (d) Stroke
      (2) Impaired mucociliary transport
         (a) Smoking
         (b) Viral or mycoplasmal infection
         (c) COPD
      (3) Chronic underlying disease
         (a) Hepatic or renal failure
         (b) Diabetes mellitus
         (c) CHF
      (4) Impaired immunity
         (a) AIDS
         (b) Chemotherapy
         (c) Alcoholism

(d) Cystic fibrosis
(e) Malnutrition
(f) Sickle cell disease, splenectomy
(g) Congenital immune deficiencies

(5) Underlying lung pathology
(a) Bronchial obstruction (foreign body, tumor)
(b) Pulmonary embolus, contusion
(c) Atelectasis

(6) Chest wall dysfunction
(a) Neuropathies or myopathies
(b) Postoperative pain
(c) Chest trauma

(7) Mechanical bypass of normal defense mechanisms
(a) Endotracheal tube
(b) Nasogastric tube
(c) Chest tube
(d) Bronchoscopy

(8) Altered upper respiratory tract flora
(a) Recent antibiotic therapy
(b) Hospitalization

e. Defense mechanisms
(1) Cough and gag reflex prevents gross aspiration.
(2) Tracheobronchial cilia remove particles >5 μm.
(3) Alveolar macrophages remove particles <5 μm.
(4) Surfactant, complement IgG and IgA limit bacterial growth.

2. Diagnostic evaluation
a. WBC count (a normal count does not exclude pathology)
(1) In a young normally healthy patient, a markedly increased WBC count (>15,000/mm^3) should prompt consideration of pneumonia as a diagnosis.
(2) In an elderly or debilitated patient, the WBC count may be increased, normal, or decreased (even if there is an associated sepsis); the only clue may be a left shift.
(3) A very high or very low WBC count is associated with increased mortality.

b. Chest radiographs
(1) Certain patterns suggest, but do not prove, a specific organism
(a) *Streptococcus pneumoniae* (most common cause of lobar pneumonia)
i. Single lobar infiltrate
ii. Small pleural effusion
iii. Abscess formation
(b) *Haemophilus influenzae*
i. Patchy (frequently basilar) infiltrates
ii. Occasional pleural effusion

(c) *Klebsiella pneumoniae*
  i. Upper lobe infiltrates
  ii. Abscess formation
(d) *Staphylococcus aureus*
  i. Patchy, multicentric infiltrates
  ii. Abscess formation
  iii. Empyema
  iv. Pneumothorax
(e) *E coli:* patchy, bilateral lower lobe infiltrates
(f) *Pseudomonas aeruginosa*
  i. Patchy, mid or lower lobe infiltrates
  ii. Abscess formation
(g) Group A streptococci
  i. Patchy, multilobar infiltrates (usually lower)
  ii. Large pleural effusion

(2) When infiltrates (patchy or lobar) are identified, always scan the radiographs for other associated findings.
  (a) Pneumothorax
  (b) Pleural effusion
  (c) Abscess formation

(3) Although CT scan may show pneumonia earlier, the radiation exposure makes it less desirable than a plain chest radiograph.

(4) Leukopenic or dehydrated patients may have normal-appearing radiographs.

(5) Other diagnoses to consider when pneumonitis is present on a chest radiograph
  (a) Pulmonary infarction
  (b) Pulmonary edema
  (c) Metastatic cancer
  (d) Pleural thickening
  (e) Parenchymal scarring
  (f) Atelectasis

c. Pulse oximetry/arterial blood gases: in patients with pneumonia, the lungs may be adequately perfused but gas exchange at the alveolus is poor, resulting in hypoxia.

d. Sputum analysis

(1) Expectorated sputum has a limited role in diagnosing the microbiologic organism involved because of contamination with oropharyngeal flora. Sputum Gram-stain results are helpful in making therapeutic decisions in only about one-third of patients.

(2) Fiberoptic bronchoscopy is the standard invasive procedure of choice for seriously ill or immunocompromised patients.

(3) Smears for acid-fast bacilli should be done in patients at risk of tuberculosis (immigrants, patients with AIDS, IV drug abusers).

e. Blood cultures

(1) A CMS core competency requirement to be ordered on all ICU patients for pneumonia

(2) Should be performed before antibiotic administration

(3) To be ordered only for patients who are seriously ill, as well as for those with presumed bacteremia, comorbid disease, immunosuppression, or rigors

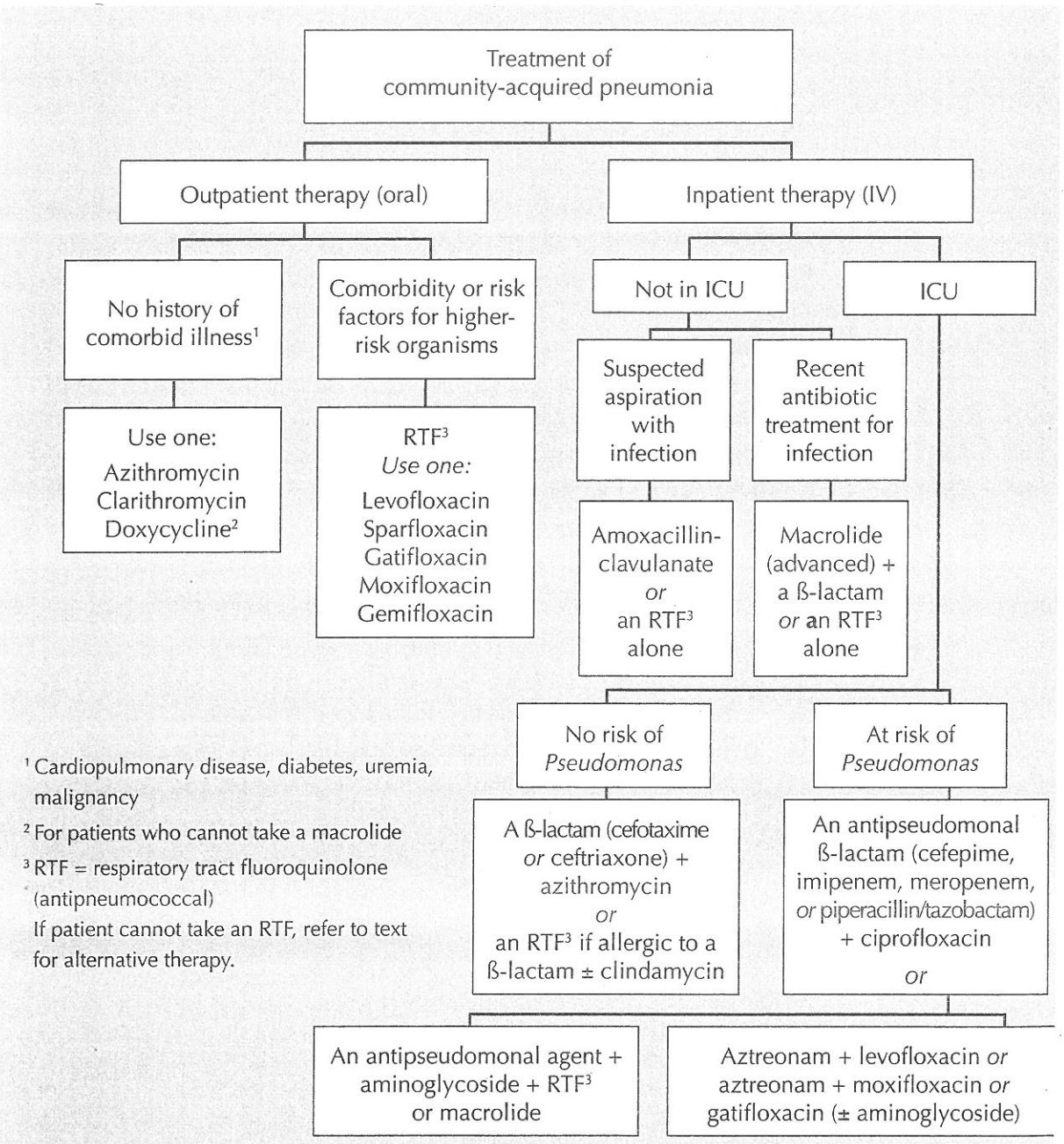
(4) "Routine" cultures on patients with pneumonia are discouraged because of a low yield of clinically useful data.

f. Pleural fluid aspiration, although not generally an emergency department procedure, helpful in excluding empyema and, in the case of a large pleural effusion, drainage c also reduce respiratory embarrassment. With smaller effusions, determining the gluc and pH of the fluid is helpful in determining treatment.

(1) pH >7.2 or glucose >60 mg/dL → antibiotics

(2) pH <7.2 or glucose <60 mg/dL → chest tube drainage with antibiotics

3. Treatment



Treatment of Community-Acquired Pneumonia

a. For inpatients with one of the following conditions requiring general medical admission:
   (1) Suspected aspiration with infection → amoxicillin-clavulanate or clindamycin
   (2) Recent antibiotic therapy for another condition → azithromycin or clarithromycin plus a ß-lactam or a respiratory fluoroquinolone alone (the regimen selected depends on the nature of the recent antibiotic therapy)

b. For ICU patients in whom:
   (1) *Pseudomonas* infection is not an issue → a ß-lactam and an advanced macrolide (azithromycin, clarithromycin) or a respiratory fluoroquinolone
   (2) *Pseudomonas* infection is not an issue, but the patient has a ß-lactam allergy → a respiratory fluoroquinolone (with or without clindamycin)
   (3) *Pseudomonas* infection is an issue →
      (a) An antipseudomonal agent (piperacillin, imipenem, meropenem, cefepime, or piperacillin-tazobactam) and ciprofloxacin or
      (b) An antipseudomonal agent and an aminoglycoside and a respiratory fluoroquinolone or a macrolide
   (4) *Pseudomonas* infection is an issue but the patient has a ß-lactam allergy →
      (a) Aztreonam and levofloxacin or
      (b) Aztreonam and moxifloxacin or gatifloxacin (with or without an aminoglycoside)

4. Types of pneumonia
   a. Pneumococcal pneumonia
      (1) Etiology
         (a) Caused by *S pneumoniae*, a gram-positive lancet-shaped, encapsulated diplococcus
         (b) At least 83 serotypes have been isolated.
      (2) Most common cause of community-acquired bacterial pneumonia and the number one cause of bacterial pneumonia in HIV-infected patients
         (a) Seen in 1 in 500 persons annually
         (b) Peak incidence in winter/early spring
         (c) Mortality rate is <5% if treated, but up to 30% if left untreated.
         (d) Pneumococcal vaccine has changed the epidemiology of this disease; the most common strain in 2009 was type 19A, which is resistant to ceftriaxone. Still, of all *S pneumoniae* infections, >90% are sensitive to ceftriaxone.
      (3) Classic clinical scenario: The patient appears acutely ill and can usually report exactly when the illness began (abrupt onset). The presence of tachypnea and tachycardia is coupled with sharp pleuritic chest pain associated with marked splinting on the affected side. There may be a history of a single, acute shaking chill, followed by a cough productive of a rust-colored sputum. Flank or back pain, anorexia, and vomiting are additional symptoms. On physical examination, the skin may be cyanotic or jaundiced, auscultation reveals crackles in the involved region, and there are signs of pulmonary consolidation (bronchial breath sounds, egophony, increased tactile and vocal fremitus). A pleural friction rub may be heard.
      (4) Diagnostic evaluation
         (a) WBC count 12,000–25,000/mm^3 but may be higher

(b) Chest radiograph
  i. Single lobar infiltrate (patchy in infants and the elderly)
  ii. Occasionally bulging fissures
  iii. Pleural effusion (25%)
(c) Sputum
  i. Gram stain reveals a single predominant gram-positive organism in pairs or chains.
  ii. Sputum culture is positive in 50%, and blood cultures are positive in 30% of cases.

(5) Management
(a) Despite the prevalence of increasingly resistant strains (up to 40%), penicillin is still the drug of choice.
(b) A macrolide or doxycycline is preferred for uncomplicated infections in outpatients.
(c) Fluoroquinolones are no longer recommended for empiric outpatient therapy in otherwise healthy patients; they are reserved for outpatients with comorbidities (COPD, diabetes, renal failure, CHF, or malignancies) and for those who have recently received antibiotics for another infection.
(d) For those patients requiring IV therapy, one of the following protocols is recommended:
  i. Cefotaxime or ceftriaxone and a macrolide (regimen preferred by the CDC) *or*
  ii. Monotherapy with an extended-spectrum fluoroquinolone

(6) Complications
(a) Sepsis
(b) Meningitis
(c) Endocarditis/pericarditis
(d) CHF
(e) Empyema (<20%)
(f) Peritonitis
(g) Septic arthritis

(7) Prognosis
(a) *S pneumoniae* types 1and 3 carry a poor prognosis
(b) Patients with any of the following also have a poor prognosis:
  i. Multilobular involvement
  ii. Leukopenia (WBC count <5,000/mm^3) or bacteremia
  iii. Jaundice
  iv. Sickle cell anemia
  v. COPD
  vi. CHF
  vii. Diabetes
  viii. Alcoholism
  ix. Splenectomy

b. *Haemophilus* pneumonia
   (1) Etiology
      (a) Caused by *H influenzae*, a gram-negative pleomorphic rod that exists in both encapsulated and unencapsulated forms
      (b) Both forms cause pneumonia, but only the encapsulated form produces bacteremia on a regular basis.
      (c) The capsular forms are separated into 6 serotypes (a–f); type b causes 95% of all human infections.
   (2) Epidemiology
      (a) The second most common cause of community-acquired bacterial pneumonia in adults
      (b) Especially common among patients with COPD and AIDS
      (c) Peak incidence is in winter/early spring.
   (3) Classic clinical scenario: The patient is elderly, debilitated, and may be diabetic, alcoholic, or both. If he or she has COPD, ask if the cough and sputum production have been getting worse. The patient typically complains of fever, shortness of breath, and pleuritic-type chest pain. On chest auscultation, there are rales but no signs of consolidation.
   (4) Diagnostic evaluation
      (a) Chest radiograph reveals patchy alveolar infiltrates and occasionally a pleural effusion.
      (b) The organism is frequently overlooked on Gram stain.
   (5) Treatment
      (a) Effective agents
         i. Amoxicillin-clavulanate
         ii. Cephalosporins (second- or third-generation)
         iii. Azithromycin (the preferred macrolide)
         iv. TMP-SMX
         v. Tetracycline or doxycycline
         vi. Fluoroquinolones (levofloxacin, sparfloxacin)
      (b) When patients require IV therapy, a second- or third-generation cephalosporin (cefuroxime, ceftriaxone) should be used.
   (6) Complications
      (a) Sepsis
      (b) Meningitis
      (c) Empyema
      (d) Arthritis

c. *Klebsiella* pneumonia
   (1) Etiology
      (a) Caused by *K pneumoniae*, a short, plump, encapsulated gram-negative bacillus that is seen in pairs
      (b) On a poor-quality Gram stain, *Klebsiella* is easily confused with pneumococcus.

(2) Epidemiology: most common in patients with
  (a) Alcoholism
  (b) Diabetes
  (c) COPD
(3) Classic clinical scenario: sudden onset of cough followed by multiple shaking chills and shortness of breath. The patient (usually a middle-aged or older man) complains of pleuritic-type chest pain and is cyanotic. Signs of pulmonary consolidation are present on physical examination.
(4) Diagnostic evaluation
  (a) Leukocytosis (75% of cases)
  (b) Chest radiograph
    i. Necrotizing right upper lobe infiltrate or abscess formation with an air–fluid level
    ii. Perihilar and patchy infiltrates are occasionally seen.
    iii. Bulging minor fissure (35%)
  (c) Gross sputum examination
    i. Dark brown, tenacious, and occasionally blood-stained
    ii. Resembles currant jelly
(5) Management
  (a) Attentive airway management because the sputum is frequently so thick that clearance is difficult
  (b) IV cephalosporin (ceftriaxone, cefuroxime, or cefotaxime) plus an aminoglycoside (gentamicin, tobramycin, or amikacin) are the initial agents of choice. Alternative agents include aztreonam and imipenem.
(6) Complications
  (a) Empyema within 24–48 hours (20%)
  (b) Sepsis
  (c) Pneumothorax

d. Other gram-negative pneumonias (occur rarely)
(1) Etiology
  (a) *E coli*
  (b) *Pseudomonas*
  (c) *Enterobacter*
  (d) *Serratia*
(2) Clinical presentation: usually immunosuppressed and debilitated patients
(3) Treatment: piperacillin/tazobactam

e. Staphylococcal pneumonia
(1) Etiology: large gram-positive cocci in pairs and clusters
(2) Epidemiology
  (a) 1% of all bacterial pneumonias
  (b) Peak incidence is during measles and flu epidemics.
  (c) Affects IV drug abusers, nursing-home patients, the debilitated, and patients recovering from influenza infection

(3) Classic clinical scenario: The patient presents with fever, multiple chills, and pleuritic chest pain. A previous course of flu was followed by the insidious onset of a cough productive of purulent sputum. Coarse rhonchi and rales are heard on chest auscultation; signs of consolidation are rare.

(4) Diagnostic evaluation

(a) WBC count usually >15,000/mm^3

(b) Chest radiograph

i. A patchy infiltrate that is initially multicentric or peripheral (because it results from hematogenous spread) ultimately progresses to lobar consolidation and abscess formation.

ii. Empyema is common.

iii. A pleural effusion may also occur.

(c) Blood cultures are usually negative unless the pulmonary involvement is metastatic.

(5) Management

(a) IV oxacillin or nafcillin are the antibiotics of choice.

(b) IV vancomycin is the alternative agent; it is usually reserved for patients who are allergic to (or resistant to) penicillin.

f. Group A streptococcal pneumonia (rare)

(1) Etiology: gram-positive cocci in pairs or chains

(2) Classic clinical scenario: sudden onset of fever, chills, and productive cough. The sputum is usually bloody and purulent; chest examination reveals fine rales without signs of consolidation.

(3) Diagnostic evaluation: chest radiograph shows multilobular bronchial infiltrates with a large pleural effusion.

(4) Management: Because of the high mortality associated with this rapidly progressive pneumonitis, patients generally require admission for IV antibiotics

(a) Aqueous penicillin is the drug of choice.

(b) A cephalosporin (ceftriaxone) or erythromycin is an alternative agent.

5. Patient assessment and risk stratification: the Pneumonia Severity Index (*ACEP Clinical Policy:* Management and Risk Stratification of Community-Acquired Pneumonia in Adults in the Emergency Department)

a. There are five severity classes based on mortality rate.

(1) Classes I, II, and III: <1% mortality

(2) Class IV: 10% mortality

(3) Class V: 27% mortality

b. Patients in Class I are <50 years old with:

(1) No comorbid conditions

(2) Normal (or near-normal) vital signs

(3) Normal mental status examination

c. Patients in Classes II–V are determined by a point system.

(1) Age in years –10 for women

(2) Nursing-home patient +10

(3) Coexisting illness

(a) Neoplastic +30
(b) Liver +20
(c) CHF +10
(d) Stroke/transient ischemic attack +10
(e) Kidney +10

(4) Physical examination
(a) Altered mental status +20
(b) Respiratory rate ≥30 breaths per minute +20
(c) Systolic blood pressure <90 mmHg +20
(d) Temperature <35°C or ≥40°C +15
(e) Pulse ≥125 beats per minute +10

(5) Laboratory studies and radiographs
(a) Arterial pH <7.35 +30
(b) BUN ≥30 mg/dL +20
(c) Na^+ <130 mEq/L +20
(d) Glucose >250 mg/dL +10
(e) Hematocrit <30% +10
(f) pO_2 <60 mmHg or O_2 sat <90% +10
(g) Bilateral pleural effusion +10

(6) Classification based on scoring
(a) Class I: <51 points
(b) Class II: 51–70 points
(c) Class III: 71–90 points
(d) Class IV: 91–130 points
(e) Class V: >130 points

(7) Disposition
(a) The Pneumonia Severity Index score may assist with decisions for admission. Admission is recommended in all patients with preexisting conditions that compromise the safety of home care or those with mitigating factors (eg, inability to take oral medications, frailty, social or psychological problems, unstable living situation, homelessness, etc).
(b) CURB-65: Tool designed to help stratify patients with community-acquired pneumonia into low, intermediate, or high risk of mortality. It is a modificati of an assessment system developed by the British Thoracic Society called CURB.

Confusion
Urea >7 mmol/L, BUN >20 mg/dL
Respiratory rate >29 breaths per minute
Blood pressure (systolic <90 mmHg, diastolic <61 mmHg)

Table 9: Calculation of CURB-65 Score

Clinical Feature of CURB-65	Points
Confusion (disoriented to time/person/place, mental test score <9), must be new	1
Uremia (BUN >19 mg/dL)	1
Respiratory rate >29 breaths per minute	1
Blood pressure (systolic <90 mmHg, diastolic <61 mmHg)	1
Age ≥65 years	1

Table 10: Management Options Based on CURB-65 Score

Score	Group	Management Options
0–1	Low mortality (1.5%)	Low risk, home therapy
2	Intermediate mortality (9.2%)	Consider hospitalization
≥3	High-risk mortality (22%)	Consider ICU

(c) SMART COP: A tool for predicting which patients with community-acquired pneumonia are likely to require intensive respiratory or vasopressor support.

Table 11: SMART-COP

Community-Acquired Pneumonia Confirmed on Chest Radiograph		
		Points
S	Systolic blood pressure <90 mmHg	2
M	Multilobar involvement on chest radiograph	1
A	Albumin <3.5 mg/dL	1
R	Respiratory rate – age-adjusted cut-offs Age ≤50 years, ≥25 breaths per minute Age >50 years, ≥30 breaths per minute	1
T	Tachycardia >125 beats per minute	1
C	Confusion (new onset)	1
O	Oxygen low – age-adjusted cut-offs Age ≤50 years: PaO_2 <70 mmHg or SaO_2 ≤93% (or if on O_2, PaO_2/FiO_2 <333) Age >50 years, PaO_2 <60 mmHg or SaO_2 ≤90% (or if on O_2, PaO_2/FiO_2 <250)	2
P	pH <7.35	2

Interpretation	
Points	
0–2	Low risk of needing intensive respiratory or vasopressor support
3–4	Moderate risk (1 in 8) of needing intensive respiratory or vasopressor support
5–6	High risk (1 in 3) of needing intensive respiratory or vasopressor support
≥7	Very high risk (2 in 3) of needing intensive respiratory or vasopressor support

Source: Charles PG, Wolfe R, Whitby M, et al. SMART-COP: A tool for predicting the need for intensive respiratory or vasopressor support in community-acquired pneumonia. *Clin Infect Dis*. 2008 Aug 1;47(3):380. By permission of Oxford University Press.

B. Atypical pneumonia

1. General characteristics
   a. Insidious onset of headache, myalgias, moderate fever, and nonproductive cough
   b. Many WBCs but no predominant organism on Gram stain
   c. WBC count normal or only moderately increased
   d. Interstitial infiltrates (usually bilateral)
   e. Protracted clinical course
2. *Mycoplasma* pneumonia
   a. Epidemiology
      (1) Most common cause of atypical "walking" pneumonia
      (2) Accounts for 10%–20% of community-acquired pneumonia
      (3) Most commonly affects children ≥3 years old and adults <40 years old (uncommon and often underdiagnosed in older adults)
      (4) Incubation period 1–3 weeks

b. Clinical presentation
   (1) Most patients have fever, chills, headache, malaise, sore throat, a dry cough, and pleuritic chest pain.
      (a) Upper respiratory infection (50%)
      (b) Earache (33%)
      (c) Anorexia, nausea, vomiting, and diarrhea in the first week (12%–14%)
   (2) Most patients have segmental rales and rhonchi on physical examination. Other findings include:
      (a) Conjunctivitis
      (b) Pharyngitis
      (c) Bullous myringitis (3%–10%)
c. Complications
   (1) Aseptic meningitis or encephalitis
   (2) Hemolytic anemia
   (3) Glomerulonephritis
   (4) Guillain-Barré syndrome
   (5) CHF, chest pain, and other cardiac abnormalities, eg, pericarditis, myocarditis, AV block
   (6) Splenomegaly
   (7) Erythema multiforme
d. Diagnostic evaluation
   (1) WBC count is usually normal or moderately increased.
   (2) Chest radiograph
      (a) One or more segmental infiltrates, dense consolidation, or a generalized interstitial pattern may be present.
      (b) Segmental (or patchy) infiltrates are usually in the lower lobes and appear as streaks radiating from the hilum.
      (c) Interstitial pneumonia characterized by a reticulonodular pattern is often associated with deterioration of pulmonary function and can progress to respiratory failure.
      (d) Small pleural effusions are present in 20% of patients.
      (e) The hallmark of this disease is the disparity between the patient's clinically benign appearance and the extensive radiographic findings.
   (3) Cold agglutinin titers are increased in up to 60% of these patients and are supportive of the diagnosis but are neither sensitive nor specific.
   (4) A 4-fold increase in complement-fixing antibody titers is diagnostic; an initial titer >1:64 is very suggestive.
e. Treatment
   (1) Erythromycin, clarithromycin, and azithromycin are the drugs of choice.
   (2) Fluoroquinolones and doxycycline are alternative agents.

3. Chlamydial pneumonia (6% of community-acquired pneumonia)
   a. Etiology: *Chlamydia pneumonia*, an obligate intracellular, gram-negative organism
   b. Epidemiology

(1) Common cause of atypical pneumonia in young adults
(2) Spread is from person to person by droplet transmission.
(3) Outbreaks generally occur as a cluster of cases in enclosed populations (eg, boarding schools, army barracks, prisons).

c. Classic clinical scenario: The patient is usually a young adult who complains of a dry cough and low-grade fever that was preceded by a sore throat. Other complaints may include laryngitis/hoarseness (present in one-third of patients), mild headache, myalgias, and diarrhea. Examination reveals rales or rhonchi (and sometimes wheezin on auscultation of the lungs and a nonexudative pharyngitis.

d. Diagnostic evaluation
(1) WBC count is usually normal.
(2) Chest radiograph typically reveals a subsegmental pneumonitis.
(3) Nasopharyngeal culture or serology confirms the diagnosis.

e. Treatment
(1) Tetracycline, doxycycline, and erythromycin are the agents of choice; a 3-week course of therapy is recommended.
(2) Azithromycin or clarithromycin (and quinolones) are also effective.

4. Psittacosis
a. Etiology: *Chlamydia psittaci*
(1) An obligate intracellular, gram-negative organism harbored in avian species
(2) Transmitted by inhalation of infected dust or droplets

b. Epidemiology: owners of pet birds (particularly parrots), pet-shop employees, poultry workers, and veterinarians most commonly affected

c. Clinical presentation
(1) Hyperpyrexia (up to 105°F [40.6°C])
(2) Severe headache (often the major complaint)
(3) Cough (occasionally associated with hemoptysis)
(4) Hepatosplenomegaly
(5) A flu-like syndrome consisting of malaise, myalgias, and an upper respiratory infection
(6) Relative bradycardia

d. Diagnostic evaluation
(1) Leukopenia (25%)
(2) Proteinuria
(3) Abnormal liver profile (high enzymes)
(4) Patchy perihilar or lower lung field infiltrates on chest radiograph
(5) Increased complement fixation antibody titer
(a) A 4-fold rise (1:32) is diagnostic.
(b) A 1:16 titer is presumptive evidence.
(c) False-positives are seen in patients with brucellosis and Q fever.

e. Treatment
(1) Doxycycline is the drug of choice; a 2-week course is recommended.
(2) Azithromycin and clarithromycin are alternative agents.

f. Complications
(1) Multiple and severe without antibiotic therapy
(2) Include hepatitis, myocarditis, endocarditis, and meningitis

5. Q fever pneumonia
a. Etiology
(1) *Coxiella burnetii*, an obligate intracellular rickettsia
(2) Highly infectious; can survive in dried soil or excrement up to 18 months and in tap water or milk for up to 42 months
b. Epidemiology
(1) People usually become infected by inhaling dust contaminated with excreta, placenta, or uterine excretions of infected sheep, goats, cattle, or parturient cats.
(2) Slaughterhouse workers, dairy farmers, and those who work closely with animals (especially farm livestock) are most commonly affected.
c. Classic clinical scenario: The patient looks ill and is diaphoretic and febrile. The usual history is sudden onset of shaking chills, high fever, myalgias, severe headache, and a nonproductive cough. Chest findings are frequently minimal (fine rales) or absent. Hepatomegaly, if present, is a clinical clue.
d. Diagnostic evaluation
(1) Abnormal liver function studies (85%)
(2) Proteinuria (62%)
(3) Sterile pyuria (12%)
(4) Rounded segmental densities in the lower lobes or lobar consolidation on chest radiograph
(5) Serologic studies are the diagnostic test of choice.
e. Treatment
(1) Early antibiotic therapy helps promote a quick response and prevents relapse or chronic Q fever.
(2) Tetracycline, doxycycline, or chloramphenicol
f. Complications associated with a prolonged illness
(1) Relapse despite antibiotic therapy
(2) Endocarditis
(3) Hepatitis
(4) Meningitis

6. Tularemia
a. Etiology: *Francisella tularensis*, a gram-negative, nonmotile, pleomorphic coccobacillus that is harbored principally in hard ticks and wild rabbits
b. Epidemiology
(1) Transmission is usually via direct contact with tissues or body fluids of infected animals, exposure to an infected tick, or inhalation of contaminated dust or water aerosol.
(2) Hunters, trappers, butchers, cooks, and campers are most commonly affected.
c. Clinical forms
(1) Ulceroglandular (most common): characterized by an indurated skin lesion at the site of inoculation and regional lymphadenopathy

(2) Typhoidal: characterized by fever, chills, weight loss, and hepatosplenomegaly
(3) Glandular
(4) Oculoglandular
(5) Oropharyngeal

d. Tularemia pneumonia: usually acquired via inhalation of contaminated aerosol or from bacteremia but may also arise as a complication of either the ulceroglandular or typhoidal forms

e. Classic clinical scenario of tularemia pneumonia: The patient (often a male) presents with high fever (104°–106°F [40°–41°C]), shaking chills, and cough (usually nonproductive). Other symptoms include chest pain, shortness of breath, and hemoptysis. Examination of the chest is often normal but may reveal rales, consolidation, or a pleural rub. Hepatosplenomegaly and a maculopapular rash may also be present. Chest radiograph usually reveals bilateral, patchy, poorly defined or ovoid infiltrates, as well as hilar lymphadenopathy and pleural effusion.

f. Diagnostic evaluation: serologic studies (ELISA) confirm the diagnosis.
(1) A 4-fold rise between acute and convalescent titers is diagnostic.
(2) A single convalescent titer ≥1:160 is very suggestive.

g. Management
(1) Streptomycin is the drug of choice.
(2) Gentamicin and kanamycin are alternative agents.
(3) Tetracycline and chloramphenicol are also effective but are associated with a high rate of relapse and should be reserved for patients who cannot tolerate any of the above agents.
(4) A live, attenuated vaccine is available for high-risk laboratory personnel.

h. Complications
(1) Mortality rate is 5%–30% without antibiotic therapy and <1% with antibiotic therapy.
(2) The course may be prolonged (up to several months).

C. Viral pneumonia

1. Common cause of community-acquired pneumonia; underlying bacterial pneumonia should be considered in patients ill enough to require admission.
2. Types of infecting viruses
   a. Respiratory syncytial virus
      (1) The most common cause of pneumonia in children <6 months old and those 3–5 years old; the elderly and the immunocompromised are also at risk of infection, as are hospitalized patients (and staff).
      (2) Seen most commonly in the winter months
      (3) Clinical presentation: fever, cough, and coryza
      (4) Diagnostic evaluation
         (a) Chest radiograph: hyperexpansion of the lungs and patchy bronchial infiltrates
         (b) Respiratory syncytial virus antigen detection tests are unreliable in adults and not recommended.
      (5) Management
         (a) Primarily supportive; if admission is required, specific therapy may be indicated.
         (b) Infants should receive a trial of ß-adrenergic therapy during the first 24 hours.

b. Parainfluenza
   (1) Second most common cause of pneumonia in children
   (2) Also causes croup and bronchitis
c. Adenoviruses
   (1) Target populations: children and military recruits
   (2) Clinical presentation: fever, cough, rhinitis, conjunctivitis, and pharyngitis
   (3) Diagnostic evaluation: chest radiographs show lower lobe infiltrates.
d. Varicella zoster virus
   (1) Occurs primarily in adults; especially severe in pregnant patients
   (2) Clinical presentation
      (a) Illness begins with a rash that is followed within a week by fever and cough associated with tachypnea and dyspnea.
      (b) 20%–40% of patients also develop cyanosis, hemoptysis, and pleuritic chest pain.
   (3) Diagnostic evaluation
      (a) Sputum analysis may reveal multinucleated giant cells.
      (b) Chest radiograph usually reveals an interstitial pneumonia; however, micronodular and lobar patterns may also be seen.
   (4) Management
      (a) This is a serious adult illness and requires admission.
      (b) Administration of IV acyclovir is indicated.
e. Influenza viruses (most common cause of viral pneumonia in adults)
   (1) Occurs between November and April
   (2) Clinical presentation
      (a) Usual syndrome has an incubation period of 1–5 days followed 2 weeks later by fever, headache, and a nonproductive cough.
      (b) Patients may have an associated bacterial pneumonia.
      (c) Up to 40% of patients with a normal chest radiograph have rales, rhonchi, and wheezing on examination.
      (d) Pure influenza pneumonia (no associated bacterial infection) is much more deadly. The elderly, pregnant patients, and those with chronic health conditions (eg, heart disease, COPD, diabetes mellitus) are at risk.
         i. Sudden weakness is followed by dyspnea, cyanosis, and ARDS.
         ii. Chest radiographs show bilateral interstitial infiltrates.
   (3) Diagnostic evaluation: rapid antigen detection assay is recommended for epidemiologic reasons.
   (4) Management
      (a) Primarily supportive
         i. Begin antiviral therapy if onset of symptoms within 48 hours or at any time if a high-risk population.
         ii. Zanamivir or oseltamivir is effective for A and B strains.
      (b) Amantadine (or rimantadine) is helpful in the treatment and prophylaxis of patients with influenza A.

f. Cytomegalovirus (CMV)
   (1) CMV pneumonia is a complication in transplant recipients and patients with advanced AIDS.
   (2) Clinical presentation
      (a) CMV can be either a true pathogen or a coexistent organism.
      (b) In transplant recipients, CMV is a true pathogen. It generally produces pneumonia within 1–3 months after transplantation and is the most common cause of death in recipients of bone marrow transplants.
   (3) Classic clinical scenario: In AIDS patients, CMV is often found in association with other pathogens and may represent a coexistent organism rather than a true pathogen. Thus, a definitive diagnosis of CMV pneumonitis in these patients requires all of the following:
      (a) A compatible clinical picture (fever, hypoxia, and infiltrates on chest radiograph)
      (b) Positive cultures for CMV
      (c) Absence of other pathogenic organisms
   (4) Diagnostic evaluation: chest radiograph most often reveals bilateral interstitial (or reticulonodular) infiltrates that begin in the periphery of the lower lobes and spread centrally and superiorly.
   (5) Management
      (a) IV ganciclovir or foscarnet
      (b) Immunoglobulin is combined with the above regimen in bone marrow recipients (who are particularly susceptible to CMV infection).

g. Hantavirus
   (1) Infection results from inhalation of aerosols that contain material contaminated with rodent urine and feces.
   (2) Epidemiology: residents of the southwestern United States (New Mexico, Arizona, Colorado, Utah) are most commonly affected.
   (3) Clinical presentation: an initial prodrome of fever, myalgia, and malaise progresses over several days and evolves into a syndrome of severe respiratory distress and shock.
   (4) Diagnostic evaluation: chest radiograph reveals bilateral interstitial infiltrates (most prominent in dependent lobes).
   (5) Management: supportive care and IV ribavirin (experimental)

D. Types of pneumonia: classic clinical scenario and etiology

Table 12: Classic Clinical Scenario and Etiology of Pneumonia

Classic Clinical Scenario	Cause/Type
Watery diarrhea, relative bradycardia, high temperature	*Legionella*
Elderly, post influenza, nursing home, cocci in clusters	*Staphylococcus*
Young, healthy male, 5-day prodrome (upper respiratory infection), respiratory failure (ARDS)	Hanta virus
Young, bullous myringitis, chest radiograph worse than clinical picture	*Mycoplasma*
COPD, gram-negative pleomorphic rods	*Haemophilus influenzae*
Alcoholic, bulging fissures, currant jelly sputum	*Klebsiella*
Elderly, recent upper respiratory infection, winter months, sudden prostration	Influenza pneumonia
Military recruits, fever, cough, rhinitis, conjunctivitis	Adenovirus pneumonia
Rusty sputum, single shaking chill, pleuritic chest pain	*Streptococcus pneumoniae*
Staccato cough	*Chlamydia*
Transplant patient 1–3 months after transplant with cough, hypoxia	CMV pneumonia
IV drug abuser, fungal infection throat, CD4	*Pneumocystis jiroveci* pneumonia
Hunter, trapper, butcher, cook, high fever	Tularemia pneumonia
Bird (parrot) owner, relative bradycardia	Psittacosis pneumonia
Slaughterhouse worker, goat, sheep, parturient cats	Q fever pneumonia

II. TUBERCULOSIS (TB)

A. Epidemiology

1. TB causes more deaths worldwide than any other single infectious agent; one-third of the world's population is infected with *Mycobacterium tuberculosis*. There are 9 million new cases per year, and 1.7 million deaths per year (2009).
2. The incidence of TB in the United States had been declining for decades, but this trend reversed in the mid-1980s; from 1985–1992, the number of cases of TB increased dramatically and in epidemic proportions. The incidence of multidrug-resistant strains of TB also increased, in large part due to noncompliance with drug therapy. Since 1993, however, the incidence has been steadily declining, with an all-time low reached in 2000. In 2008, the incidence of TB was 4.2/100,000 cases in the United States.
3. Circumstances that contributed to the resurgence in the mid 1980s included the HIV epidemic; congregate living in nursing homes, prisons, and shelters; immigration; a decline in the ability of cities and states to maintain TB control programs; and other social factors such as substance abuse, poverty, and homelessness. Of these factors, the HIV epidemic is probably the single most significant influence. Comprehensive strengthening of control activities has reduced the rate of transmission.

4. TB is an AIDS-defining opportunistic infection and is the only opportunistic infection in AIDS patients that is transmitted by the respiratory route to both immunocompromised and immunocompetent hosts.

B. Pathophysiology

1. The causative organism is *Mycobacterium tuberculosis*, a weakly gram-positive obligate aerobic rod with acid-fast staining properties that multiplies very slowly (once every 12–24 hours).
2. Transmission almost always occurs via aerosolized droplets produced by coughing, sneezing, talking, or breathing. Infection develops when these contaminated droplets are inhaled and reach the alveoli.
3. Once in the alveoli, tubercle bacilli are phagocytized (but not killed) by alveolar macrophages and proliferate within these cells to form a primary focus of infection (primary TB), usually in the lower lobes. Organisms may also spread from this initial site of infection through the lymphatics to regional lymph nodes and to distant organs via the bloodstream. This bacillemia is usually asymptomatic, but it produces metastatic foci throughout the body that may become active later in life. These foci are preferentially established in areas of high oxygen tension such as the apical and posterior segments of the upper lobes of the lung, the kidneys, bones, and brain.
4. Most infected patients mount an effective immune response and have no further infectious sequelae. T lymphocytes reach sufficient numbers to control the infection 2–10 weeks (average 6–8 weeks) after exposure. The tuberculin skin test becomes positive at this time, indicating that cell-mediated immunity has developed. Immunocompromised patients, however, may be unable to mount an adequate immune response. In these individuals, a rapidly progressive primary infection resulting in early death can evolve.
5. After a period of dormancy, some infected patients go on to develop active disease. This generally occurs when the patient's immune response is altered in some manner.
   a. The lifetime risk of reactivation in the general population is 10% but is much greater in patients with impaired cellular immunity.
   b. Conditions associated with an increased rate of conversion to active disease
      (1) AIDS (rate of progression is 7%–10% per year)
      (2) Immunosuppressive therapy, including steroids
      (3) Renal failure, hemodialysis
      (4) Diabetes
      (5) Malnutrition, alcoholism
      (6) Malignant disease
      (7) Postgastrectomy and postintestinal bypass states
      (8) Transplant recipients

C. Clinical presentation

1. Pulmonary TB (inactive, or dormant, foci)
   a. Asymptomatic in 90% of patients; can be identified only by development of a positive TB skin test and possibly a Ghon complex on chest radiograph
   b. A pneumonitis may also be seen (usually in the lower lobes).
2. Reactivation TB (endogenous reactivation of dormant foci)
   a. Most common clinical form of TB and is seen most often in the elderly
   b. Symptoms include low-grade fever, night sweats, malaise, weight loss, and productive cough (most common symptom).

c. Signs of chronic wasting are present in most patients.
d. Sites of involvement include the apical and posterior segments of the upper lungs, kidneys, bones/joints, and brain.
   (1) Pulmonary involvement is present in >80% of patients.
   (2) Extrapulmonary involvement is present in 15% of the general population but is greater in patients with HIV.

3. Pulmonary TB (active foci)
   a. Clinical onset
      (1) Insidious: patients (usually debilitated) present with a chronic cough (most common symptom) and constitutional symptoms of reactivation such as malaise, weight loss, and fever. As the cough progresses over time, it becomes productive of mucopurulent sputum and is often associated with hemoptysis. Patients may also complain of a dull ache or tightness in the chest.
      (2) Abrupt: some patients present with acute onset of fever, chills, cough (most common symptom), and myalgias that mimic an episode of acute bronchitis or pneumonia. Unless TB is considered in the differential diagnosis and smears of sputum for acid-fast bacilli are obtained, these patients may be misdiagnosed as having bacterial pneumonia.
   b. Chest examination is often unremarkable but may reveal rales or consolidation in the presence of extensive pulmonary involvement.

4. Extrapulmonary TB can result from primary infection or reactivation and may involve almost any organ in the body. It may also take a disseminated form (miliary TB). The signs and symptoms produced are determined by the structures that are affected.
   a. TB meningitis (most rapidly progressive form of TB)
      (1) Results from seeding during the primary infection or from rupture of a subependymal lesion (Rich foci) into the subarachnoid space.
      (2) Clinical onsets
         (a) Insidious: patients often present with a nonspecific febrile illness of 1–6 weeks duration, followed by intermittent headache, confusion, personality changes, stiff neck, diplopia, photophobia, cranial nerve palsies, decreased level of consciousness, and seizures.
         (b) Fulminant: some patients, particularly children, may present acutely with fever and delirium in association with a severe headache and stiff neck.
      (3) Diagnostic testing
         (a) Positive TB skin test (75%)
         (b) Cerebrospinal fluid analysis (acid-fast stain not usually positive)
            i. ↑ Pressure and protein
            ii. ↓ Glucose
            iii. ↑ WBC count of 100–1,000/mL (predominantly lymphocytes)
   b. Pleural TB
      (1) Results from rupture of a parenchymal focus into the pleural space
      (2) Associated pleural effusion is exudative in nature with laboratory evaluation revealing:
         (a) Increased protein
         (b) Low pH

(c) Normal or low glucose
(d) WBC count of 1,000–5,000/mL (mostly monocytes)
(e) An acid-fast smear that is often negative
(3) Pleural biopsy is helpful in making the diagnosis.

c. Genitourinary TB
(1) Patients usually present with urinary symptoms (dysuria, frequency) hematuria, or flank pain. Constitutional symptoms may also be present.
(2) Urinalysis reveals:
(a) Pyuria without bacteriuria
(b) Low pH

d. Miliary (disseminated) TB
(1) A multisystemic process resulting from a progressive primary infection (immunocompromised patients) or from secondary bloodstream seeding during recrudescence of previously dormant foci
(2) Clinical presentation
(a) Symptoms are usually nonspecific and may include fever, anorexia, weight loss, and weakness. Depending on the sites of involvement, more specific symptoms (eg, dyspnea, cough, headache) may also be present.
(b) Physical findings may include:
i. Fever
ii. Pulmonary findings
iii. Hepatomegaly
iv. Lymphadenopathy
v. Splenomegaly
vi. Tubercles of the retina (circumscribed, spheroid, granulomatous lesions with three distinct zones, the only specific finding)
(3) Diagnostic evaluation
(a) Anemia and WBC abnormalities (leukopenia, leukemoid reactions, agranulocytosis)
(b) Hyponatremia
(c) Negative TB skin test 25%–50% of the time (especially in the elderly and immunocompromised)
(d) Chest radiograph typically reveals small nodular densities that are uniformly distributed throughout both lung fields. A pleural effusion may also be present.

5. TB and HIV
a. TB is an AIDS-defining illness and generally produces disease at an earlier stage of HIV infection than other opportunistic infections.
b. The incidence of TB in HIV-infected patients approaches 60% and currently represents the greatest health care risk to the general public from the HIV epidemic.
c. Co-infection with TB and HIV results in:
(1) A greater incidence of extrapulmonary disease
(2) More atypical clinical findings
(3) An increased incidence of tuberculin nonreactivity and negative acid-fast bacilli smears

(4) A greater frequency of unusual and atypical chest radiographs
(5) Decreased cavitary disease
(6) More antibiotic resistance
(7) Higher relapse and mortality rates
(8) A larger number of adverse drug reactions and multidrugresistant TB (especially to rifampin and isoniazid)

D. Diagnostic evaluation

1. TB must be considered in the differential diagnosis of any patient who presents with respiratory complaints or extrapulmonary symptoms, particularly if the patient is a member of a high-risk group (patients with AIDS, immigrants, IV drug abusers, residents/employees of longterm care facilities).
2. Tests used to establish the diagnosis include the TB skin test, chest radiograph, and microbiologic studies for acid-fast bacilli.
3. TB skin test
   a. The standard test for detecting infection with *M tuberculosis*
   b. Involves intradermal administration of purified protein derivative and is read 48–72 hours after administration
   c. A positive reaction indicates the presence of infection but not necessarily the presence of active disease (must be confirmed by culture).
   d. Criteria for interpreting the test as true-positive vary with patient background as follows:
      (1) 5 mm induration is positive in patients with:
         (a) Known or suspected HIV infection
         (b) An abnormal chest radiograph
         (c) Close contact with a person with active TB
      (2) ≥10 mm induration is positive in:
         (a) Residents of or employees in high-risk settings, such as health care facilities, homeless shelters, and prisons
         (b) IV drug abusers
         (c) Immigrants from an area with a high incidence of TB
         (d) Certain high-risk minority groups (Hispanics, black Americans, Native Americans)
      (3) ≥15 mm induration is positive in all others
   e. A negative test does not exclude the diagnosis, because some patients (particularly those who are immunocompromised) are anergic.
   f. A false-positive test may be due to infection with *M avium* or *M kansasii* (nontuberculous mycobacteria). Clues to this diagnosis include the following:
      (1) No history of risk factors for TB
      (2) A negative skin test (or a reaction smaller than a true-positive)
      (3) History of COPD
4. Chest radiograph
   a. Primary TB
      (1) Small parenchymal infiltrates located in any area of the lung and unilateral hilar adenopathy
      (2) These lesions may subsequently calcify to form a Ghon complex.

(3) Inflammatory infiltrates of the lower lobes with associated hilar adenopathy are seen in patients with progressive primary infections and clinically evident disease.

(4) Hilar adenopathy is the radiologic hallmark of primary TB in children.

b. Reactivation/pulmonary TB

(1) Typically an upper lobe process. Nodular densities are most often seen in the apical (Simon foci) or posterior segments of the upper lobe but may also be found in the upper segment of the lower lobe.

(2) Associated cavitation may or may not be present.

c. Miliary (disseminated) TB

(1) May initially be normal but classically reveals small nodules (1–3 mm) scattered throughout both lung fields in a miliary pattern.

(2) A pleural effusion (frequently unilateral) may also be present.

d. HIV-infected patients

(1) The chest radiograph is often atypical and may even be normal.

(2) Upper lobe cavitary lesions are rare, while hilar or mediastinal adenopathy and lower lobe infiltrates are more common.

(3) A diffuse interstitial pattern that is easily mistaken for Pneumocystis jerovici pneumonia may also be seen.

5. Microbiologic studies

a. Staining of sputum for acid-fast bacilli with Ziehl-Neelsen or fluorescent (fluorochrome) staining (which is more sensitive)

(1) Provides a rapid presumptive diagnosis of TB; number of bacilli seen correlates with the degree of infectivity.

(2) Positive smears have a specificity of 98%.

(3) Smear results should be confirmed by culture.

b. Culture of sputum or tissue for acid-fast bacilli

(1) More sensitive than staining and is the "gold standard" for confirming the diagnosis of TB

(2) Although traditional cultures take 3–6 weeks, new radiometric techniques can confirm the diagnosis in as few as 5 days; DNA probes, reverse transcription, and polymerase chain reaction tests allow for identification of TB in a matter of hours but, because of technical problems, have not received approval for routine clinical use.

E. Management

1. As soon as the diagnosis is suspected, the patient should wear a mask and be placed in an isolation room.
2. Because of the emergence of multidrug-resistant strains of TB, it is now recommended that initial therapy for TB include four drugs until susceptibility tests are available.
3. The initial drug regimen of choice is isoniazid, rifampin, pyrazinamide, and streptomycin or ethambutol; in the absence of drug resistance, isoniazid and rifampin taken for 9 months is curative. (Preventive drug therapy also includes isoniazid [ie, for those with a positive intradermal skin test].)
4. Adverse effects associated with these drugs:

a. Isoniazid

(1) Multiple neurologic entities, including peripheral neuritis; pyridoxine (vitamin B_6) is administered concurrently to prevent isoniazid-induced neuropathy.

(2) Hepatitis
(3) Hypersensitivity reactions
(4) Drug-induced interactions with ketoconazole and fluconazole

b. Rifampin
(1) Hepatitis
(2) Thrombocytopenia
(3) Drug interactions with coumadin, oral contraceptives, digitalis derivatives, methadone, dapsone, cyclosporin, corticosteroids, oral hypoglycemic agents, ketoconazole, and fluconazole → decreased blood levels and effectiveness
(4) Orange-colored tears, saliva, and urine

c. Pyrazinamide
(1) Hyperuricemia
(2) Hepatitis
(3) Arthralgias
(4) Rash

d. Ethambutol
(1) Optic neuritis
(2) Rash

e. Streptomycin
(1) Vestibular nerve damage
(2) Nephrotoxicity

5. Before starting therapy with these agents, baseline studies should be obtained.
a. Liver function tests, BUN/creatinine, and CBC with platelet count for all patients
b. Visual acuity (and red-green color perception) for patients being treated with ethambutol
c. Serum uric acid for patients being treated with pyrazinamide

III. PLEURAL EFFUSION

A. Definition

1. An abnormally large collection of fluid within the pleural space, reflecting the presence of an underlying intrathoracic or extrathoracic disease process
2. Types of effusions
a. Transudates: excessive hydrostatic pressure (eg, CHF) or insufficient oncotic pressure (↓ serum protein) → low protein plasma infiltrates
b. Exudates: lymphatic blockage due to malignancy or pleural capillary damage due to infectious disease → high protein plasma infiltrates

B. Clinical presentation

1. Pleuritic pain is common with infection. Other associated symptoms include fever, cough, and shortness of breath.
2. Physical examination findings
a. Splinting ± a pleural friction rub → pleurisy

b. Dullness to percussion + ↓ breath sounds + ↓ tactile fremitus → pleural fluid
c. Bronchial breath sounds + egophony → atelectasis
d. Normal breath sounds + distended neck veins + left parasternal lift + accentuated P_2 on cardiac examination → massive pulmonary embolism

C. Diagnostic evaluation

1. Chest radiographs
   a. Small effusions are most easily detected on a lateral decubitus film with the affected side down; accumulations of 5–50 mL of fluid can be detected with this view.
   b. Small effusions can be missed entirely on supine films and are generally not apparent on PA and lateral films until ≥200 mL of fluid is present.
   c. Early signs of small effusion
      (1) PA view → faint obscuring of the costophrenic angle
      (2) Lateral view → blunting or loss of the costophrenic angle
      (3) Lateral decubitus view → fluid layers out
   d. Signs of a moderate effusion
      (1) Ground-glass appearance of lung fields
      (2) No air bronchograms
   e. Sign of a large effusion → opacification (partial or complete)
   f. Pleural fluid in a fissure → a "phantom" or "pseudotumor"
2. Ultrasound
   a. Small effusions can be easily detected with point-of-care ultrasound.
   b. Anechoic space seen on ultrasound image
      (1) Complicated effusions may show septations and hypoechoic material.
      (2) Important to identify surrounding structures of diaphragm and atelectic lung tissue
3. Thoracentesis: the definitive procedure
   a. Contraindications
      (1) Uncooperative patient
      (2) Coughing or hiccups
      (3) Local skin infection
      (4) Coagulopathies
      (5) Anticoagulant therapy
   b. Technique
      (1) Mark the site with ultrasound.
      (2) The posterior entry is the primary approach. Insert the needle *immediately above* the rib to avoid the neurovascular bundle.
      (3) Loculated effusions may require CT-guided needle aspiration.
      (4) Withdrawing fluid
         (a) Reexpansion pulmonary edema may occur if fluid is removed too rapidly.
         (b) Limit removal of fluid to ~2 L.
      (5) After the procedure, obtain a chest radiograph to exclude an iatrogenic pneumothorax.

c. Diagnostic evaluation
   (1) CBC with differential
   (2) Serum protein, lactate dehydrogenase (LDH), amylase, and glucose (<40 mg/dL → empyema)
   (3) Cytology studies
   (4) Gram stain and smear for acid-fast bacilli
   (5) Cultures for aerobes, anaerobes, mycobacteria, and fungi
   (6) pH (<7.2 suggests empyema; <6.0 suggests esophageal rupture)

d. Order serum protein and LDH levels, because the criteria for classifying pleural fluid as an exudate or transudate includes the protein and LDH pleural fluid to serum ratios.

e. Light criteria to determine exudate versus transudate: if any one of the following is met, the fluid is exudative:
   (1) Pleural fluid protein/serum protein >0.5
   (2) Pleural fluid LDH greater than two-thirds the upper limit of normal for the laboratory
   (3) Pleural fluid LDH/serum LDH >0.6

f. Differential diagnosis
   (1) Transudates
      (a) CHF (most common cause of effusions, followed by infections and malignancy, below)
      (b) Constrictive pericarditis
      (c) Hypoalbuminemia
      (d) Nephrotic syndrome
      (e) Cirrhosis
      (f) Peritoneal dialysis
   (2) Exudates
      (a) Infections
         i. Bacterial pneumonia
         ii. TB (usually primary)
      (b) Malignancy
      (c) Connective tissue and hypersensitivity disorders
         i. Rheumatoid arthritis
         ii. Systemic lupus erythematosus
         iii. Dressler syndrome
         iv. Drug-induced
      (d) Pancreatitis
      (e) Subphrenic abscess
      (f) Abdominal surgery
      (g) Uremia
      (h) Esophageal rupture
      (i) Pulmonary emboli and infarction (the most common causes of pleural exudates, along with pneumonia and malignancy)

(j) Hemothorax
(k) Superior vena cava obstruction causing venous hypertension or thoracic duct obstruction
(l) Chylothorax
(m) ARDS

D. Critical facts

1. Most common cause of pleural effusion: CHF
2. Most common cause of transudate: CHF
3. Most common cause of exudate: infection
4. Normal amount of pleural fluid: 0.1–0.2 mL/kg body weight
5. Transudate pathophysiology: increased hydrostatic pressure or decreased oncotic pressure; very little protein
6. Exudate pathophysiology: increased membrane permeability or defective lymphatic drainage; large amount of protein
7. Massive effusions are more commonly due to malignancy, but CHF can do it, too.
8. Effusions are best seen in lateral decubitus with the effusion side down.
9. Pulmonary embolism is the most commonly overlooked condition in the evaluation of patients with pleural effusion.
10. If the difference between serum albumin and pleural albumin is >1.2 g/dL, the effusion is a transudate.
11. If pH <7.3, consider pneumonia, malignancy, rheumatoid effusion, TB, systemic acidosis.
12. If pH <7.0, consider empyema.
13. Bloody effusion means traumatic tap, trauma, neoplasm, or pulmonary infarction.
14. TB is the most common cause of pleural effusion in third-world countries.

IV. ASPIRATION PNEUMONIA

A. Pathophysiology

1. Aspiration pneumonia is an inflammation of lung parenchyma precipitated by foreign material entering the tracheobronchial tree.
2. The initial pathologic changes (first few minutes) produced by fluid aspiration are nonspecific and independent of the type of fluid aspirated. These changes include collapse and expansion of individual alveoli, reflex airway closure, and interstitial edema → ventilation-perfusion mismatching and hypoxia
3. Final extent and severity of pulmonary injury, however, are determined by the specific substance aspirated and depend on three factors:
   a. pH and volume of the aspirate
   b. Presence of particulate matter (such as food)
   c. Bacterial contamination
      (1) In community-acquired aspiration pneumonia, streptococcal species are the most common aerobic isolates.
      (2) The most commonly isolated aerobes from nosocomial aspiration pneumonia are gram-negative bacilli and *Staphylococcus aureus*.

4. The pH and specific content of the aspirate affect the pathologic changes produced as follows:
   a. pH >2.5
      (1) The additional injury produced by neutral fluids is determined by the volume and content.
      (2) The larger the volume of aspirate, the greater the mortality and morbidity.
      (3) Aspiration of lipid material → a chronic granulomatous reaction lipoid pneumonia
      (4) Aspiration of fluid containing food particles → a persistent inflammatory reaction that progresses within 6 hours to hemorrhagic pneumonitis → a granulomatous reaction resembling TB
      (5) Aspiration of charcoal → bronchiolitis obliterans
   b. pH <2.5
      (1) Aspiration of fluid with a pH <2.5 produces pulmonary changes resembling those of a chemical burn.
      (2) Aspiration of as little as 0.3 mL/kg in children or 20–25 mL in adults → immediate reflex airway closure, destruction of surfactant-producing alveoli, alveolar collapse, and irreversible damage to pulmonary capillaries. Sequelae include pulmonary hemorrhage, bronchial epithelial degeneration, and pulmonary edema.
      (3) Secondary bacterial infection eventually results.
         (a) Anaerobes predominate in community-acquired aspirations.
         (b) A mixture of gram-negative aerobes and anaerobes are typically isolated in hospital-acquired aspirations.
5. Foreign-body aspiration
   a. The leading cause of accidental home death in children <6 years old
   b. Complete obstruction causes death by asphyxiation in 4–6 minutes.
   c. Peripheral aspiration causes pneumonitis and lung abscess.

B. Risk factors
1. Depression of the cough or gag reflex (general anesthesia, chronic illness, drug overdose, use of sedative medications)
2. Nasogastric tubes
3. Esophageal strictures, dysmotility, and reflux
4. Esophageal obturator airway
5. Tracheostomies
6. Poor oral hygiene

C. Clinical presentation
1. Aspiration of fluid and oropharyngeal bacteria can be silent and actually occurs to some extent in normal individuals during sleep.
2. Pathologic aspirations, however, such as those occurring during a substance abuse stupor or in a comatose state when the normal protective airway reflexes are decreased or lost, tend to produce a more devastating clinical picture.
   a. Sudden onset of coughing or choking
   b. Tachypnea, tachycardia, and cyanosis
   c. Wheezing, rales, or rhonchi

d. Large amounts of frothy, bloody sputum

e. Hypotension

3. Most patients (>90%) develop signs and symptoms within 1 hour of the event.

4. Patients who have aspirated a foreign body (and have incomplete obstruction) present with choking, a spasmodic cough, and wheezing. Examination of the chest may reveal asymmetric chest wall movement, decreased breath sounds, wheezing, and hyperresonance to percussion on the involved side.

D. Diagnostic evaluation

1. Arterial blood gases

a. The usual finding is hypoxia with respiratory alkalosis.

b. Severe aspiration respiratory failure with a combined respiratory and metabolic acidosis

2. Chest radiograph

a. Radiographic findings are often delayed.

(1) Atelectasis is the initial finding and may be seen as early as 1 hour after aspiratior

(2) Infiltrates develop 6–12 hours later and most frequently involve the right lower lo (if aspiration occurred in the upright position).

b. Foreign body aspiration → an end-expiratory PA film demonstrates a hyperexpanded lung on the involved side (usually the right side, because the right mainstem bronchu has less of an acute angle than the left).

E. Management

1. Provide supplemental oxygen.

a. High-flow oxygen by nasal cannula or face mask may be sufficient for some patients.

b. Patients who are hypercarbic (or remain hypoxic) despite these measures should be intubated and mechanically ventilated.

c. If the patient has an altered level of consciousness and a decreased gag reflex, immediate endotracheal intubation is needed.

2. Consider noninvasive positive-pressure ventilation (NIPPV); if little or no improvement in respiratory distress or hypoxemia while on NIPPV after 30–60 minutes, intubation wil likely be required.

3. Bronchoscopy is useful in removing large particles and clearing the large airways in larg volume aspirations. Bronchial irrigation with saline solution, however, should be avoide it has no beneficial effect and may be harmful.

4. Antibiotics should be reserved for elderly and chronically ill patients, as well as for thos who develop clinical evidence of infection (fever, purulent sputum, leukocytosis). Bacte aspiration pneumonia occurs over a period of several days in >60% of cases of chemica aspiration. Antibiotic therapy is based on the origin of the infection.

a. Community-acquired aspiration pneumonia → empiric treatment with a third- or fourth-generation cephalosporin or a fluoroquinolone (with antipneumococcal coverage) or a ß-lactam with a ß-lactamase inhibitor and a macrolide

b. Nosocomial aspiration pneumonia → empiric treatment with piperacillin/clavulanat or a fluoroquinolone (with antipneumococcal coverage) and clindamycin (If an absc is present, clindamycin has excellent penetration.)

5. Supportive care measures
   a. Humidified oxygen
   b. Bronchodilators
   c. Chest physiotherapy

F. Complications
1. Acute respiratory failure
2. Severe sepsis
3. Pneumonia, empyema, and lung abscess
4. Pulmonary fibrosis (for chronic aspiration)

G. Mortality rate
1. Varies with the pH and contamination of the aspirate
2. 40%–70% if the pH is <2.5; almost 100% if the pH is <1.8 or the aspirate is grossly contaminated, ie, Boerhaave syndrome

H. Prevention of aspiration in the emergency department
1. Any patient with a depressed or absent gag reflex should be intubated (preferentially with rapid-sequence intubation) using a high-volume, low-pressure, cuffed endotracheal tube.
2. When gastric lavage is indicated in the obtunded or comatose patient, intubate first and then place the patient in Trendelenburg on his or her left side before lavage.
3. Administering metoclopramide to accelerate gastric emptying may be helpful.

V. LUNG ABSCESS

A. Definition and epidemiology
1. A cavitation of the lung parenchyma resulting from local suppuration and central necrosis; often precipitated by aspiration of oropharyngeal secretions
2. Predisposing factors (lead to suppression of cough/gag reflexes)
   a. Factors that suppress the cough/gag reflex (ethanol, stroke, seizures)
   b. Esophageal motility disorders, strictures, and cancer
   c. Pulmonary disorders
      (1) Pneumonia
      (2) Embolic phenomena
         (a) Pulmonary embolus with cystic infarction
         (b) Septic emboli
      (3) Vasculitis
      (4) Infected cysts
   d. Periodontal disease (anaerobic lung abscess)

B. Etiology: most lung abscesses are polymicrobial and involve either strictly anaerobes or a mixture of anaerobic organisms.
1. Anaerobic

a. *Fusobacterium*
b. *Bacteroides*
c. Streptococci (microaerophilic and anaerobic)

2. Aerobic
a. *Staphylococcus aureus* (often follows influenza in a flu epidemic)
b. *Streptococcus pneumoniae*
c. Alpha streptococci
d. *Pseudomonas*
e. *Klebsiella pneumoniae*
f. *E coli*
g. *Proteus*

4. Other organisms
a. *Mycobacterium*
b. *Histoplasma*
c. *Coccidioides*
d. Lung flukes and *Entamoeba histolytica*

C. Clinical presentation

1. Coincident with the development of cavitation (1–2 weeks after aspiration)
2. History
a. Systemic signs and symptoms: weakness, fever, weight loss
b. Pulmonary signs and symptoms: dyspnea, chest pain, and a cough productive of a fetid and bloody sputum
3. Physical examination
a. Poor dentition, gingivitis, and foul-smelling breath
b. Signs of localized consolidation or cavitation on auscultation
c. Hemoptysis

D. Diagnostic evaluation

1. CBC: increased WBC count with a left shift and anemia
2. Cavitation with an air–fluid level
a. Chest radiograph or CT scan of the chest
b. Most common sites are the posterior segment of the right upper lobe and superior segment of the left and right lower lobes.
c. Findings that suggest empyema rather than abscess
(1) An air-fluid level at the site of a previous pleural effusion
(2) A cavity with an air–fluid level that tapers at the pleural border
(3) An air–fluid level that crosses a fissure
(4) air–fluid level that extends to the lateral chest wall
3. Sputum analysis: because of oropharyngeal contamination, only bronchoscopically obtained sputum, transtracheal, or transthoracic specimens are reliable for anaerobic culture and sensitivity.

E. Management

1. Antibiotic therapy
   a. Clindamycin is currently the antibiotic of choice for an uncomplicated lung abscess. It is given IV until the patient remains afebrile for 5 days and then is continued orally for 6–8 weeks.
   b. Alternative agents include penicillin (when penicillin resistance is not a problem), metronidazole, or cefoxitin.
2. Indications for surgery
   a. Life-threatening hemoptysis
   b. Bronchopleural fistula
   c. Tumor or empyema
   d. A residual cavity

F. Late complications

1. Chronic lung abscess
2. Empyema
3. Bronchopleural fistula
4. Brain abscess

VI. EMPYEMA

A. Epidemiology

1. A collection of pus in the pleural space or fissures
2. Mechanism of formation
   a. Hematogenous or lymphatic spread from pneumonia
   b. Infection from a chest tube, thoracentesis, or thoracotomy
   c. Esophageal perforation and mediastinitis
   d. Rupture of a mediastinal lymph node
   e. Aspiration pneumonia
   f. Direct extension from retropharyngeal or subdiaphragmatic abscesses or from vertebral osteomyelitis

B. Etiology

1. *Mycobacterium tuberculosis*
2. *Staphylococcus*
3. *Pseudomonas*
4. Gram-negative organisms
5. Anaerobes

C. Clinical presentation

1. Acute illness: fever and chills with pleuritic chest pain and shortness of breath
2. Subacute illness (more common): weight loss and fatigue
3. Physical examination: decreased breath sounds, dullness on percussion, and decreased excursion of the involved hemithorax

D. Diagnostic evaluation
1. Chest radiograph: air–fluid level in the pleural space or loculated fluid
2. Thoracentesis: confirms the diagnosis

E. Clinically useful classifications for empyema
1. American Thoracic Society categorization of empyema by volume
   a. Minimal: <10 mL of free fluid
   b. Moderate: >10 mL of fluid less than half of hemithorax
   c. Large: greater than half of hemithorax
2. Fluid analysis categorization
   a. pH <7.2
   b. Glucose <60 mg/dL
3. Clinical decision making: if any of the above is true regarding the fluid analysis (empyema), chest tube is indicated. Consult cardiothoracic surgery.

F. Management
1. Pleural drainage via tube thoracostomy, image-directed catheterization, thoracoscopic drainage, or thoracotomy with open drainage and decortication
2. High-dose, broad spectrum IV antibiotics for ≥2 weeks

G. Complications
1. Empyema necessitans (dissection into the subcutaneous tissues or through the chest w
2. Bronchopleural fistula
3. Permanent loss of lung tissue

VII. HEMOPTYSIS

A. Definitions
1. **Hemoptysis**
   a. **Coughing up blood originating from the pulmonary parenchyma or tracheobronc tree**
   b. **Most often not life threatening**
   c. **However, when massive, hemoptysis can produce airway obstruction as well as hemorrhagic shock and require urgent intervention.**
2. **Massive hemoptysis**
   a. **A single expectoration >50 mL or 600 mL blood in 24 hours**
   b. Necessitates transfusion to maintain stable hematocrit

B. Etiology varies with age of patient
1. Infection/inflammation: most common cause
   a. **Bronchitis (especially chronic): most common cause**
   b. **Pneumonia (especially *Klebsiella, Staphylococcus,* or influenza virus)**
   c. **Parasites (ascariasis, schistosomiasis)**
   d. **Endocarditis**

   e. Bronchiectasis (most common causes of massive hemoptysis are bronchiectasis, tuberculosis, lung abscess, and neoplasm)
   f. Tuberculosis
   g. Lung abscess
2. Neoplasms (especially bronchogenic cancer)
3. Cardiovascular disorders
   a. Mitral stenosis
   b. CHF
   c. Pulmonary hypertension (primary) or embolism/infarction
   d. AV malformation/fistula
   e. Congenital heart disease
   f. Thoracic aortic aneurysm
   g. Pulmonary embolism
4. Trauma
5. Immunologic disorders
   a. Goodpasture syndrome
   b. Vasculitis
6. Other
   a. Cystic fibrosis
   b. Blood dyscrasias
   c. Drugs (ASA, tissue plasminogen activator, ethanol, etc)
   d. Coagulopathies
7. Idiopathic (~5%–15% of cases)
8. Iatrogenic
   a. Swan-Ganz catheter → rupture of pulmonary artery
   b. Chronic tracheostomy → tracheoinominate fistula

C. Hemoptysis versus hematemesis

1. Need to distinguish from hematemesis or blood swallowed from epistaxis via physical examinations
2. "True" hemoptysis is:
   a. Initiated and accompanied by vigorous coughing
   b. Generally bright red and foamy and does not contain particles of food

D. Clinical presentation: determined in large part by underlying cause

1. Bacterial pneumonia
   a. Small amounts of blood in sputum, productive cough, and fever
   b. Examination may reveal rales or rhonchi and signs of consolidation.
2. CHF
   a. Small amounts of blood in sputum, shortness of breath
   b. Examination may reveal rales, an S_3 gallop, jugular venous distention, hepatomegaly, ascites, or peripheral edema.

3. Bronchogenic carcinoma
   a. Generally have chronic cough and weight loss
   b. Examination may reveal signs of consolidation and clubbing.
4. Tuberculosis
   a. May be massive hemoptysis
   b. Patients are often cachectic and have a chronic cough.
   c. Examination may reveal posttussive rales.

E. Diagnostic evaluation

1. The severity and suspected etiology of the hemoptysis determine the extent of the evaluation performed in the emergency department setting.
2. Chest radiograph
   a. Should be obtained in all patients and may reveal signs of underlying pulmonary or cardiovascular disease
   b. 50% are normal.
3. CBC: establishes a baseline hematocrit
4. Prothrombin time (INR)/partial thromboplastin time and platelet count: useful in patients who are on anticoagulants to exclude blood dyscrasias
5. Arterial blood gases: indicated in patients with respiratory distress or massive hemoptysi
6. Type and crossmatch: should be obtained in all patients with massive hemoptysis
7. Sputum: should be sent for Gram and acid-fast stains, culture (for bacteria, fungi, and mycobacteria), and cytologic examination
8. Bronchoscopy
   a. Useful both diagnostically and therapeutically
   b. Should be obtained on an emergent basis in patients with massive hemoptysis
9. Selective arteriography
   a. Can be used to localize and embolize the site of bleeding in patients with massive hemoptysis
   b. Particularly useful when the site is peripheral to the bronchoscope's field of view
10. High-resolution chest CT: usually reserved for patients in whom the chest radiograph is normal and bronchoscopy is unrevealing

F. Management: determined by the volume of hemoptysis and underlying condition

1. Minimal hemoptysis
   a. Treatment is aimed at correcting the underlying problem.
   b. For example, patients with pneumonia should receive antibiotics.
   c. Specific therapy for the hemoptysis itself is generally unnecessary.
2. Massive hemoptysis
   a. Initial therapy is aimed at maintaining the airway, stabilizing the patient, and terminating the bleeding.
   b. Intubate the patient: mainstem intubation may be helpful if able to isolate nonbleeding hemithorax.
   c. Place the patient in Trendelenburg with the bleeding side down (to protect the uninvolved lung and maximize gas exchange).
   d. Suction as needed.

e. Establish two large-bore IV lines and replace blood loss rapidly.
f. Obtain immediate consult with pulmonologist and thoracic surgeon.
g. Consider interventional radiology early for embolization.
h. Perform bronchoscopy to identify source of bleeding.
i. Once the patient is stabilized and bleeding has been controlled, further evaluation and treatment of the underlying pathology can be done.

VIII. PNEUMOTHORAX

A. Spontaneous pneumothorax

1. A collection of air in the pleural space (in the absence of trauma) that is divided into primary and secondary forms
2. Primary (idiopathic) spontaneous pneumothorax
   a. Occurs in healthy individuals who lack evidence of underlying pulmonary pathology; however, most are smokers. There may also be a recent history of vigorous exercise.
   b. Results from rupture of a subpleural bleb (often apical) or a weak pleural segment → air leaking into the pleural space → pulmonary collapse
   c. Causative factors are not always present but may include:
      (1) Atmospheric pressure changes (eg, scuba diving, fighter pilots)
      (2) Performance of the Valsalva maneuver in association with abuse of marijuana/cocaine or injection into the central venous system (the "pocket shot") by IV drug users
      (3) Marfan syndrome
      (4) Cigarette smoking
      (5) Family history of pneumothorax
   d. Incidence is greatest in young adults 20–40 years old.
   e. More common in males than females (male to female ratio is 5:1); tall, thin males who smoke are most commonly affected.
   f. Onset is usually during rest or sleep.
   g. Recurrence rate is 20%–50% over the following 2–5 years.
3. Secondary spontaneous pneumothorax
   a. Occurs in individuals with underlying lung pathology that damages the alveolar/pleural barrier or causes an increase in intrabronchial pressures
   b. Associated medical conditions
      (1) Airway disease
         (a) COPD (most common cause)
         (b) Chronic bronchitis
         (c) Asthma
         (d) Cystic fibrosis
      (2) Infection
         (a) Pneumonia, particularly *Pneumocystis jerovici* pneumonia in AIDS patients
         (b) Tuberculosis

(3) Neoplasms
(4) Interstitial lung disease
(a) Collagen vascular disease
(b) Pneumoconioses
(c) Sarcoidosis
(d) Idiopathic pulmonary fibrosis
(5) Other
(a) Toxic drugs, especially aerosolized pentamidine
(b) Chemical and radiation pneumonitis
(c) Smoking
(d) Endometriosis ("catamenial" pneumothorax)

c. Individuals >40 years old are most commonly affected.

4. Iatrogenic pneumothorax: occurs in association with performance of procedures such as:
a. Subclavian vein catheterization and CPR (most common causes in emergency department)
b. Endotracheal intubation (increased frequency with mainstem intubation)
c. Intercostal nerve block
d. Percutaneous lung biopsy
e. Thoracentesis
f. Bronchoscopy (with transbronchial biopsy)
g. CT-guided thoracic procedures

B. Classic clinical scenario

1. The patient presents with sudden onset of pleuritic chest pain and dyspnea. Mild tachycardia and tachypnea may also be present. Your first thought might be pulmonary embolism, especially if the patient is a middle-aged woman with risk factors for thromboembolic disease. The chest pain is usually anterior but may radiate into the neck, back, or ipsilateral shoulder. An associated cough and subcutaneous emphysema of the neck and chest are occasionally present.
2. Classic physical findings are decreased breath sounds, decreased tactile fremitus, and hyperresonance to percussion on the affected side. However, these signs may be subtle or absent in patients with COPD or small pneumothoraces.
3. On ECG, ST segment changes and T-wave inversion may be found, thus mimicking cardiac ischemia.

C. Diagnostic evaluation

1. Chest radiographs
a. Findings on a standard PA chest film (taken in inspiration)
(1) A fine line (the edge of the collapsed lung) running parallel to the chest wall but separated from it by a space
(2) Absence of lung markings along the lung periphery in the space beyond this line
b. If the standard chest radiograph does not reveal a suspected pneumothorax, a supine film may be helpful (especially in the hypotensive patient; upright films are not advised in these patients).

(1) In this position, a pneumothorax will collect along the costophrenic sulcus (rather than along the apex of the lung in an upright position), thus making a small pneumothorax more apparent.

(2) Chest radiographs taken in full expiration have not been shown to enhance detection of pneumothorax.

c. Radiologic findings that may be confused with pneumothorax

(1) Skin folds

(2) Tubing outlines

(3) Clothing

(4) Bullae or cysts

2. Ultrasound for diagnosis (curvilinear probe)

a. Loss of pleural sliding

(1) Shallow probe depth

(2) Decrease gain.

(3) Minimize any image processing setting; look for artifacts.

(4) Use zoom setting to view pleural line.

(5) Identify "bat sign" of two ribs with pleural sliding between rib cross-section.

b. Absence of B lines/lung rockets

c. M-mode demonstrates stratosphere or barcode sign; seashore sign present with normal lung sliding

d. Lung point (site at which the visceral and parietal pleura separate) may be visible.

D. Management

1. Observation, simple catheter aspiration, and tube thoracostomy
2. Approach selected is determined by the size of the pneumothorax, the degree of symptomatology, the presence of underlying pulmonary pathology, the reliability of the patient, and whether the patient has had prior pneumothoraces.
3. All patients should be placed on supplemental oxygen, because it hastens resolution of the pneumothorax.
4. Observation (inpatient or outpatient)

a. Acceptable if the pneumothorax is small (<15%–20%), and the patient is healthy, reliable, and minimally symptomatic

b. When this option is chosen, serial chest radiographs must be performed to exclude progressive accumulation of air within the pleural cavity.

c. One approach is to observe the patient in the emergency department for 6 hours and then repeat the chest radiograph. If the chest radiograph remains unchanged, the patient may be discharged to home with instructions to return for a repeat chest radiograph in 24 hours (or sooner if symptoms progress).

d. Reabsorption occurs at a rate of 1.25% of the volume of intrapleural air per day and is hastened by the administration of 100% oxygen.

5. Simple catheter aspiration

a. Reduces a large or moderate pneumothorax to a small one, which can then be left to resorb on its own

b. Appropriate for mildly to moderately symptomatic patients with primary spontaneous or needle-induced pneumothoraces

c. A chest radiograph should be performed immediately after the aspiration (and a hours later) to verify that aspiration was successful and that reaccumulation ha not occurred.

6. Tube thoracostomy
   a. The therapeutic "gold standard"
   b. Considered to be mandatory in the presence of:
      (1) Significant underlying pulmonary pathology
      (2) An expanding pneumothorax
      (3) A pneumothorax >25%
      (4) Bilateral or tension pneumothorax
      (5) Trauma
      (6) Significant dyspnea
      (7) Detectable pleural fluid
      (8) Positive-pressure ventilation
      (9) Previous contralateral pneumothorax

E. Tension pneumothorax

1. A life-threatening complication
2. Evolution: air enters the pleural space on inspiration but cannot escape on expirat (ball-valve effect) → progressive accumulation of air within the pleural cavity and total collapse of the affected lung → a shift of mediastinal structures to the opposi hemithorax → compression of the contralateral lung and impairment of venous re → decrease in cardiac output and development of signs of shock
3. Classic clinical scenario: The patient is hypotensive, cyanotic, and in severe respira distress. Air hunger develops, and the respiratory rate increases. The patient becor agitated and restless and displays decreasing mental activity. The trachea is deviate to the contralateral side (even significant tracheal deviation may be difficult to det clinically), and there is hyperresonance to percussion and absent breath sounds or involved side. Jugular venous distention may be present. When the diagnosis is ma even suspected, the positive intrapleural pressure must be released immediately; d wait for radiograph confirmation. Insert a large-bore needle (14 gauge) anteriorly involved hemithorax through the second or third intercostal space midclavicular li This allows time for a tube thoracostomy to be performed.

IX. ASTHMA

A. Definition and epidemiology

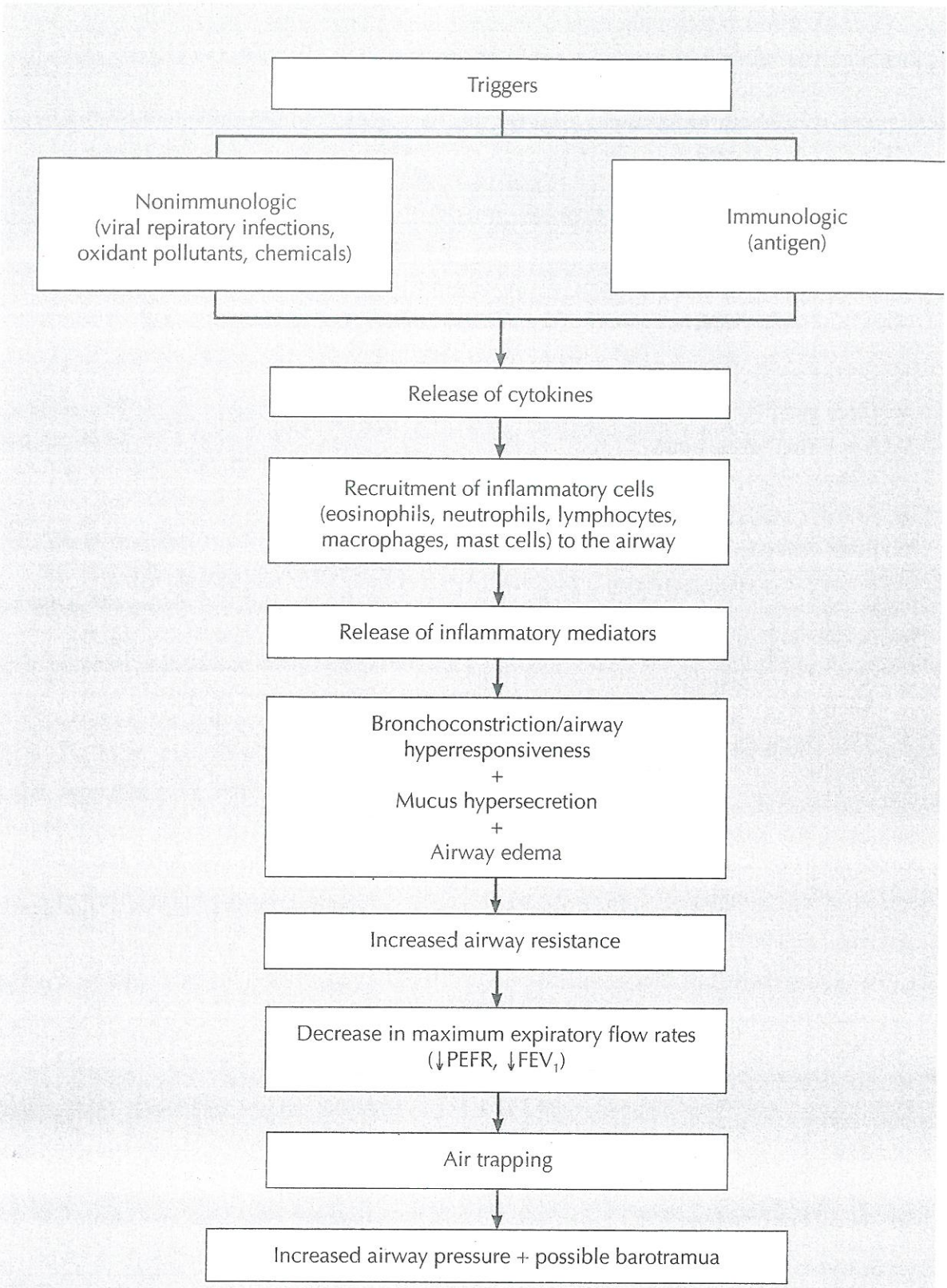
1. A chronic lung disorder characterized by:
   a. Increased airway responsiveness (an exaggerated bronchoconstriction response variety of stimuli
   b. Airway inflammation
   c. Reversible airway obstruction
2. Can occur at any age but is more common in children and adolescents; 50% of pa develop asthma before age 10.

3. Prevalence is increasing.
   a. Affects 22 million Americans
   b. Most common chronic disease of childhood
4. Asthma-related morbidity and mortality rates have been climbing over the last 20 years, especially in children <6 years old.
5. Most common predictors of fatal asthma seem to be a past history of intubation/mechanical ventilation, ICU admission, and recent history of poorly controlled asthma.

B. Etiology

1. In the past, asthma was classified as either intrinsic (not IgE-mediated) or extrinsic/allergic (atopic or IgE-mediated). These distinctions, however, no longer reflect current understanding. Asthma is now classified as intermittent, mild persistent, moderate persistent, or severe persistent. Classification is based on:
   a. Daily symptoms
   b. Nocturnal awakenings
   c. Frequency of use of short-acting ß-agonist
   d. Activity limitations
   e. Lung capacity volumes
2. Asthma is viewed as a state of bronchial hyperreactivity with multiple potential "triggers." An immunologic reaction mediated by IgE is only one such "trigger," and it can occur in both atopic and nonatopic individuals.
3. Triggers that can initiate an asthmatic response
   a. Allergens
   b. Viral respiratory infections (may be the most common cause)
   c. Sinusitis
   d. Exercise
   e. Inhaled irritants
   f. Strong odors
   g. Medications (NSAIDs, ASA, ß-blockers) and food additives (MSG, sulfites, some dyes)
   h. Gastroesophageal reflux
   i. Cold exposure and changes in humidity
   j. Strong emotions (via vagal efferent pathways)
   k. Endocrine factors (menses, pregnancy, thyroid disease)
4. Conditions that may mimic asthma
   a. Gastroesophageal reflux disease
   b. CHF
   c. Upper airway obstruction
   d. Vocal cord dysfunction

C. Pathophysiology



Asthma Triggers

D. Clinical presentation

1. Symptoms usually awaken the patient at night.
   a. Cough
      (1) May be dry or productive of sputum
      (2) May be the only finding in patients with cough-variant asthma
   b. Dyspnea
   c. Wheezing
   d. Chest tightness
2. Typical examination findings
   a. Tachypnea
   b. Tachycardia
   c. Wheezing: Although the most common manifestation of asthma, wheezing is not an accurate indicator of the severity of an attack; it may be absent in patients with severe obstruction. Also remember that not all that wheezes is asthma.
   d. Prolonged expiratory phase
   e. Hyperresonance to percussion

3 Findings suggestive of severe airway obstruction
   a. Dyspnea so severe the patient is able to speak only a few words at a time, and air movement on auscultation of the lungs is poor.
   b. Use of accessory muscles (sternocleidomastoid, scalenus) reflects diaphragmatic fatigue.
   c. Heart rate >120 beats per minute
   d. Respiratory rate >30 breaths per minute
   e. Pulsus paradoxus ≥12 mmHg (defined as ≥12 mmHg drop in systolic blood pressure during inspiration) implies that the FEV_1 is less than half the normal predicted FEV_1 for that patient and reflects the degree of airway obstruction, air-trapping, and ventilatory effort.
   f. Silent chest indicates that airflow is dramatically reduced and is no longer adequate to promote wheezing.
   g. Diaphoresis
   h. Inability to recline on the stretcher
   i. Paradoxical respirations herald impending respiratory failure.
   j. Cyanosis is a late finding appearing just before respiratory arrest.
   k. Altered mental status (confusion, agitation, somnolence) may indicate hypercapnia and often reflects impending respiratory failure.

E. Differential diagnosis

1. Pulmonary embolus: most of these patients have one or more risk factors for deep venous thrombosis/pulmonary embolism. Order spiral CT with contrast if suspected (V/Q scans may be indeterminant in patients with acute asthma); angiography is indicated if the CT is negative, but clinical suspicion is high.
2. CHF (cardiac asthma): distinguishing features include older age, bibasilar rales, an S_3 gallop, pink frothy sputum, and jugular venous distention; ß-natriuretic hormone assays may be useful in distinguishing the two.

3. Upper airway obstruction (cancer, laryngeal edema, foreign body inhalation): remember that these patients usually have stridor (not wheezing); a patient with wheezing usually has lower airway obstruction. If in doubt, listen to tracheal breath sounds; any abnormality indicates upper airway obstruction.
4. Carcinoid tumors are typically associated with postprandial flushing and GI upset.
5. Chronic bronchitis or COPD with acute exacerbation: these patients are generally older, have a history of smoking (along with a chronic productive cough), and no true symptom-free intervals.
6. Eosinophilic pneumonias and invasive worm infestations
7. Endobronchial conditions (cancer, foreign body aspiration, bronchial stenosis): wheezing is unilateral
8. Drugs (ß-blockers, ACE inhibitors)
9. Allergic or anaphylactic reactions: urticaria, angioedema, hypotension, and/or GI symptoms are clinical clues.
10. Chemical irritants, insecticides, and anticholinergics: a clue is a history of exposure in an otherwise normal individual.
11. Noncardiogenic pulmonary edema (ARDS)

F. Diagnostic evaluation

1. Pulmonary function tests (PEFR or FEV_1): Although recommended to gauge treatment efficacy and severity of the exacerbation, PEFR and FEV_1 have not proved beneficial or predictive based on multiple studies. However, they are useful in documenting a trend and serve as a comparison to baseline.
   a. Should be measured before and after each treatment with an adrenergic agent
   b. The PEFR is the easiest test to perform in the emergency department, because it is done at the bedside with a portable peak expiratory flow meter.
   c. These studies provide an objective measure of the degree of airflow obstruction present; they are useful in assessing the severity of an attack, the response to treatment, and the need for close follow-up or admission.
   d. Indicators of severe bronchospasm and probable hospital admission
      (1) PEFR <100 L/min or FEV_1 <1 L before treatment
      (2) PEFR or FEV_1 <50% baseline/predicted (adults)
      (3) Failure of PEFR to improve by ≥10% after initial treatment
      (4) PEFR <300 L/min or FEV_1 <2.1 L after aggressive treatment
2. CBC
   a. Adds little to the overall assessment of the patient; not routinely warranted
   b. Mild eosinophilia is frequently present, reflecting the asthmatic condition.
   c. The WBC count is often increased from the stress of an asthma attack or chronic steroid use, but it may also be increased in the setting of pneumonia.
3. Sputum
   a. Eosinophils are typically present and can make the sputum appear purulent in the absence of infection.
   b. Other findings may include:
      (1) Charcot-Leyden crystals (crystallized eosinophilic granules)

(2) Curschmann spirals (spiral casts of the airways formed from mucus and epithelial cells)

(3) Creola bodies (clusters of columnar epithelial cells)

4. Pulse oximetry
   a. Should be used to assess and follow the adequacy of oxygenation; however, a near-normal pulse oximetry may be present in a patient with pending respiratory failure due to hypercapnia; bedside capnometry may be a better way to monitor these patients.
   b. Oxygen saturation ≤90% indicates a severe asthmatic attack and significant hypoxemia.
5. Arterial blood gases
   a. Do not measure pulmonary function per se but rather the ability to exchange oxygen and carbon dioxide
      (1) Pulmonary function tests measure how well a patient is doing.
      (2) Arterial blood gases measure how poorly a patient is doing.
   b. The real value of arterial blood gases lies in the assessment of respiratory failure, ie, detecting the presence of eucapnia or hypercapnia and acidosis.
      (1) They are indicated in patients with signs of impending respiratory failure:
         (a) Fatigue or exhaustion
         (b) An altered sensorium
         (c) Worsening or failure to improve after adequate treatment
         (d) SaO_2 ≤90%
      (2) Severity of respiratory failure: arterial blood gas results
         (a) Mild respiratory failure: increased pH, decreased pCO_2, normal or decreased pO_2 (respiratory alkalosis)
         (b) Moderate respiratory failure: normal pH, normal pCO_2, decreased pO_2
         (c) Severe respiratory failure: decreased pH, normal or increased pCO_2, decreased pO_2 (respiratory acidosis)
      (3) Arterial blood gas values consistent with severe asthma
         (a) pO_2 <60 mmHg
         (b) pCO_2 >45 mmHg
         (c) pH <7.35
6. Chest radiograph
   a. May reveal evidence of hyperinflation (increased AP diameter and/or flattening of the diaphragms), increased bronchial markings, and/or atelectasis but is generally nondiagnostic
   b. Is most useful in excluding the complications of asthma and conditions that mimic it
   c. Not obtained routinely but indicated in patients who:
      (1) Are febrile
      (2) Have focal physical findings suggestive of pneumonia or barotrauma (pneumothorax, pneumomediastinum)
      (3) Do not respond to aggressive treatment
      (4) Present with their first episode of wheezing

7. ECG
   a. Unless ischemia is suspected, routine ECGs are generally not helpful diagnostically.
   b. A right ventricular strain pattern is seen in 30%–40% of patients.
8. Theophylline level should be checked in patients taking this medication.

G. Management

1. Pharmacologic agents and their mechanism of action
   a. $ß_2$-adrenergic agonists
      (1) Promote bronchodilation by increasing cyclic AMP
      (2) May also modulate mediator release from mast cells and basophils as well as promote mucociliary clearance
      (3) Primary effect is on the small peripheral airways.
      (4) Onset of action is <5 minutes.
   b. Anticholinergic agents
      (1) Promote bronchodilation by inhibiting vagally mediated bronchoconstriction, ie, competitively antagonize acetylcholine (the transmitter released by the vagus nerve) at the postganglionic parasympathetic effector cell junction → decrease in cyclic GMP → bronchodilation
      (2) Primary effect is on the large central airways.
      (3) Onset of action is delayed up to 30 minutes and peaks in 1–2 hours.
   c. Corticosteroids
      (1) Believed to produce their effects by:
         (a) Increasing the responsiveness of ß-adrenergic receptors in airway smooth muscle
         (b) Limiting recruitment and activation of inflammatory cells
         (c) Interfering with arachidonic acid metabolism and the synthesis of leukotrienes and prostaglandins
      (2) Onset of action is gradual with initial improvement occurring 3 hours after administration and peaking in 6–12 hours. Early administration (within 1 hour) results in fewer hospital admissions and a lower rate of relapse after emergency department discharge.
   d. Antileukotrienes are being used more frequently in managing chronic asthma.
2. Management of adults in the emergency department (for treatment of children, see page 653)
   a. Administer oxygen to all asthmatic patients; many are hypoxic, and treatment with ß-adrenergic agents may initially worsen hypoxia by exacerbating ventilation-perfusion mismatching. Verify the adequacy of arterial oxygen saturation by pulse oximetry, and use continuous capnometry (if available) to assess adequacy of ventilation.
   b. Begin treatment with ß-adrenergic agonists because of their rapid onset of action (<5 minutes) and effectiveness in promoting bronchodilation. Aerosolized therapy is preferred over parenteral therapy; onset of action is more rapid, and it provides comparable or superior bronchodilation, has fewer adverse effects, and avoids the need for painful injections. The oral route is not appropriate in the acute setting.
      (1) Inhaled agents: administer one of the following using a handheld nebulizer.

(a) Albuterol 0.5% solution: the preferred agent because of its $ß_2$-specificity and fewer adverse effects

i. Dosage: 2.5 mg in 2–3 mL normal saline every 20–30 minutes up to 3 doses, then hourly

ii. Continuous nebulization at 15 mg/hr for 2 hours is safe and effective in adults with severe bronchospasm; it should be considered for those patients who present with a PEFR <200 L/min or an FEV <50% of predicted.

(b) Metaproterenol sulfate 5% solution

i. Dosage: 15 mg in 2–3 mL normal saline every 20–30 minutes up to 3 doses, then hourly

ii. Some α- and ß-adrenergic effects limit its usefulness.

iii. Although numerous studies have demonstrated that delivery of ß-agonists (using a metered-dose inhaler with a spacing device) can achieve bronchodilation equivalent to that achieved by nebulization, this method requires more patient effort and is not recommended for patients with severe obstruction.

(2) Injectable (SC) medications should be reserved for those patients who are too sick to make an effective inspiratory effort; alternative medications include:

(a) Epinephrine 1:1,000 solution

i. Dosage: 0.3 mg every 20–30 minutes up to 3 doses

ii. Both α and ß effects, but the ß effects predominate.

(b) Terbutaline 1 mg/mL

i. Dosage: 0.25 mg every 20–30 minutes up to 3 doses

ii. More $ß_2$ selective than epinephrine and has a longer duration of action.

(c) These agents should be strongly considered in patients with severe asthma who have an inadequate ventilatory effort with inhaled agents; in these cases, cardiovascular disease is not a contraindication. In life-threatening situations, epinephrine can be lifesaving.

c. Anticholinergic agents

(1) For all patients who present with exacerbation of acute asthma, anticholinergics and ß-agonist therapy should be used together in initial management of an acute attack. Efficacy of anticholinergic therapy increases proportionately with the severity of the attack, but some benefit is gained even in less severe attacks.

(2) When anticholinergics are used in combination with ß-adrenergic agonists, effects are additive, and a small incremental benefit may be derived; although their effects are delayed in onset, they provide a synergistic reduction in bronchospasm and a more prolonged effect. Addition of anticholinergics to ß-agonists (and glucocorticoid) therapy improves pulmonary function in the first 90 minutes and decreases the rate of hospitalization.

(3) Nebulized ipratropium bromide 500 mcg in 2 mL normal saline every 3–4 hours is the agent of choice; it may be added to the nebulizer along with the standard dose of ß-agonist being administered. If ipratropium is being used as a first-line agent, the dose may be repeated with the first three ß-agonist inhalation treatments. This agent is an atropine derivative but has limited systemic absorption and produces far fewer adverse effects than atropine.

(4) Ipratropium is also effective in reversing bronchospasm secondary to ß-blocking agents.

d. Corticosteroids

(1) Treat the inflammatory component of asthma and should be administered early

(2) Reduce the rate of relapse and rate of hospital admission

(3) Underuse is believed to be an important cause of fatal asthma.

(4) Should be administered to the following patients:

(a) Those who show minimal or no improvement after initial sympathomimetic therapy

(b) Those who are currently taking (or have recently discontinued) a steroid preparation

(c) Those with signs of severe obstruction, eg, tachycardia >120 beats per minute, pulsus paradoxus ≥12 mmHg, PEFR or FEV_1 <40% baseline

(d) Those with a history of asthma-associated respiratory failure

(e) Those whose attack has been prolonged (these patients are more likely to have significant bronchial wall edema and mucus impaction)

(f) Those who relapse (return to the emergency department for the same asthmatic attack)

(5) Oral and IV routes are equally effective.

(6) Use one of the following agents:

(a) Methylprednisolone 60–125 mg IV: parenteral drug of choice

(b) Hydrocortisone 200–500 mg IV

(c) Methylprednisolone 60 mg orally

(d) Prednisone 1–2 mg/kg orally

(7) Patients who receive steroids in the emergency department and are subsequently discharged should be continued on oral steroids (methylprednisolone or prednisone 1 mg/kg/day) at home for a minimum of 3–5 days and a maximum of 14 days. Prolonged therapy with a gradually tapering dose is recommended for patients taking maintenance doses for >10 days.

(8) Aerosolized corticosteroids are potentially irritating (can stimulate cough or bronchospasm) and, therefore, should not be used during an acute attack.

e. Other considerations

(1) Evaluate for evidence of dehydration, and administer replacement fluids (orally or IV) as needed.

(2) Do not administer sedatives, narcotics, or tranquilizers.

(3) Avoid the use of IV isoproterenol; it is highly dysrhythmogenic and carries a risk of myocardial injury resulting from ischemia or infarction.

(4) Magnesium sulfate should be administered in moderate to severe, nonresponding acute asthma. The dosage is 2–3 g over 10 minutes and should be given while continuing inhalation therapy.

(5) Administration of helium should be considered for patients with severe asthma. It decreases the work of breathing and, therefore, allows time for medications that have already been administered to work (thereby avoiding the need for intubation, which is a problem in some asthmatic patients).

(6) Check serum electrolytes in adult patients who require >3 doses of ß-agonists, because these drugs can cause hypokalemia, hypomagnesemia, and hypophosphatemia.

(7) Reserve antibiotics for patients with evidence of infection (fever, infiltrate on chest radiograph, evidence of sinusitis); respiratory tract infections that trigger exacerbations of asthma are usually viral in etiology.

f. Noninvasive positive-pressure ventilation

(1) May be used in patients with respiratory failure (or impending failure) who are not responsive to therapy but who have normal mentation and facial anatomy

(2) Bilevel positive-airway pressure is the modality of choice for patients who are able to cooperate.

(a) Start with an inspiratory pressure of 8 cm H_2O and an expiratory pressure of 3 cm H_2O.

(b) Increase settings based on pulse oximetry and arterial blood gas results.

(c) Patients who do not improve over 30–60 minutes require intubation.

g. Intubation with assisted ventilation

(1) Mechanical ventilation in these patients can produce excessively high airway pressures and is associated with considerable morbidity and mortality.

(2) The initial goal of mechanical ventilation in these patients is not necessarily to restore the pCO_2 to normal; the respiratory rate and tidal volume required to do this can produce excessive peak inflation pressures → alveolar gas trapping and overdistention → high risk of barotrauma

(3) These patients should be intentionally hypoventilated (a technique referred to as "controlled mechanical hypoventilation" or "permissive hypercapnia"). This technique increases the amount of time available for expiration and thereby minimizes air trapping. Suggested initial ventilator settings are tidal volume 6–8 mL/kg, respiratory rate 8–10 breaths per minute, and an inspiratory flow rate of 80–100 L/min. The moderate hypercapnia that may occur with these ventilator settings is well tolerated and less dangerous than persistently high airway pressures.

(4) Volume-cycled ventilators with higher peak pressures and flow rates should be used initially to deliver an adequate tidal volume. Plateau pressure is most important in minimizing barotrauma. Aim for plateau pressure of <30.

(5) Criteria for intubation

(a) Arterial blood gas triad of progressive hypoxemia, hypercapnia, and acidosis *or*

(b) Persistent hypercapnia in spite of therapy *or*

(c) Persistently poor or worsening pulmonary function tests *or*

(d) Clinical signs of respiratory failure (cyanosis, apnea) *or*

(e) Change in mental status (confusion, somnolence) *or*

(f) Exhaustion/obtundation (patient looks at you but cannot speak)

(6) Adjuncts to intubation/mechanical ventilation

(a) Lidocaine should be used in the asthmatic patient before induction and paralysis to minimize exacerbation of bronchospasm.

(b) Ketamine can be used to provide sedation for intubation (or as an induction agent for rapid-sequence intubation) and has the benefit of inducing

bronchodilation; avoid in patients with hypertension and those at risk of coronary artery disease (may increase pulse and blood pressure).

(c) Succinylcholine is the paralytic of choice. Pancuronium is useful when muscle paralysis is required to facilitate ventilation; it does not cause histamine release.

(d) Benzodiazepines may be used when sedation is needed (it will minimize emergence of hallucinations if ketamine was used); opiates induce histamine release and should be avoided.

(e) Heliox should be considered for mechanically ventilated asthmatic patients with significant respiratory acidosis and markedly increased airway pressures; it lowers airway resistance and decreases the work of breathing. A helium–oxygen mixture of 80:20 or 70:30 (preferred) should be used.

h. Asthma in pregnancy

(1) Incidence of asthma can increase in pregnancy: one-third of patients with asthma get worse with pregnancy, one-third have no change, and one-third may get better.

(2) Respiratory alkalosis (pCO_2 of 30–35) is a normal finding in pregnancy. Do not be concerned about respiratory compromise unless the pCO_2 is >35.

(3) Management of asthma in pregnant patients is similar to that in nonpregnant patients. The goal of treatment is to ensure adequate oxygenation for both mother and fetus while avoiding (whenever possible) any drugs that may pose a risk to the fetus.

(a) All patients should receive supplemental oxygen (maternal hypoxia → impaired fetal oxygenation → fetal complications). Place the pregnant patient on her left side to minimize compression of the inferior vena cava.

(b) Inhaled ß-agonists are the first-line drugs of choice. If parenteral therapy is required, terbutaline is preferred over epinephrine; the use of SC epinephrine early in pregnancy is associated with an increased incidence of congenital malformations, and in late pregnancy with premature labor (and should be avoided).

(c) Corticosteroids are also safe in pregnancy and should be administered in all but the mildest exacerbations.

(d) Anticholinergic agents and theophylline may also be used. The clearance of theophylline in the third trimester, however, is reduced; serum levels must be carefully monitored.

(e) Asthma medications that are appropriate for administration during pregnancy are also safe for use during lactation.

X. CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

A. Definition and pathophysiology

1. Often referred to as a single disease but is actually a triad of three distinct disease processes

a. Chronic bronchitis: defined clinically as a productive cough for 3 months over 2 consecutive years, characterized by airway hypersecretion and inflammation that causes a chronic productive cough for an extended period of time

b. Emphysema: defined anatomically and characterized by irreversible enlargement of the air spaces distal to the bronchioles

c. Asthma: characterized by airway hyperreactivity and inflammation. In most COPD patients, these three entities coexist, but the contribution each makes to pulmonary dysfunction varies from individual to individual. However, some COPD patients present with predominately emphysema (pink puffers) or chronic bronchitis (blue bloaters).

2. Evolution

a. COPD begins in the early twenties and is detected only by pulmonary function testing. Early pathologic changes are completely silent.

b. As the disease progresses, signs and symptoms appear with a full-blown clinical picture as early as in the thirties in some patients.

c. Chronic bronchitis ensues with a typical history of chronic, recurrent production of excessive mucus.

d. Repeated inflammation leads to increased resistance or decreased caliber of the small bronchi and bronchioles. As alveolar hypoventilation subsequently occurs, hypoxemia and hypercarbia result. Ventilation-perfusion mismatching occurs (thus promoting hypoxemia), while increased physiologic dead space ventilation leads to alveolar hypoventilation, hypercarbia, and further hypoxemia.

e. In addition to obstruction of the peripheral airways, destruction and coalescence of the alveolar cell structure (particularly in dominantly emphysematous disease) results in reduction of the total "matched" alveolar-capillary surface area for diffusion of gases (oxygen cannot get in, CO_2 cannot get out).

f. Later on, chronic and progressive airflow obstruction can lead to right-sided cardiac strain that results in pulmonary hypertension (loud P_2 on auscultation) and cor pulmonale.

3. Fourth most common cause of death

B. Risk factors

1. Smoking (most important factor)
2. Exposure to second-hand smoke
3. Environmental pollution
4. Industrial or occupational exposure
5. α_1-antitrypsin deficiency
6. Cystic fibrosis
7. Recurrent pulmonary infections

C. Etiology of acute decompensation

1. Superimposed respiratory infection (acute bronchitis, pneumonia): the most common precipitating factor of acute COPD exacerbation. Influenza and pneumococcal vaccination recommended.
2. Changes in ambient temperature, humidity, or air pollution levels
3. Noxious environmental exposures
4. Spontaneous pneumothorax
5. Pulmonary embolus
6. Noncompliance with (or underdosing of) medications
7. Inappropriate treatment (ß-blockers, sedatives)

8. Acute CHF
9. Continued cigarette smoking

D. Clinical presentation in combined disease

1. Dyspnea is the most common complaint, followed by cough (occasionally with hemoptysis), chest tightness, and fatigue. A history of morning headache may be due to a rising $PaCO_2$ (hypercapnia).
2. Physical findings
   a. Tachypnea
   b. Prolonged expiratory phase
   c. Increased AP chest diameter ("barrel chested")
   d. Decreased breath sounds
   e. Wheezing, rales, and rhonchi
   f. Lip-pursing, accessory muscle use, cyanosis, and diaphoresis
   g. Pulsus paradoxus
   h. Distant heart sounds, a loud P_2, and right-sided CHF

E. Clinical presentation when emphysema or bronchitis predominates

1. Emphysema ("pink puffer")
   a. Dyspneic
   b. Thin, anxious, alert appearance
   c. Increased AP chest diameter (overinflation)
   d. Tachypneic and hypotensive
   e. Uses accessory muscles to breathe
   f. Decreased breath sounds, faint end-expiratory rhonchi, and hyperresonance on percussion
2. Chronic bronchitis (blue bloater)
   a. Productive "wet" cough
   b. Stocky build, polycythemic, and cyanotic
   c. Normal chest diameter
   d. Little air-hunger
   e. Subxiphoid or retrosternal heave → right ventricular hypertrophy
   f. Signs of CHF (jugular venous distention, S_3 or S_4 gallop, murmur of tricuspid insufficiency, scattered rales and rhonchi, edema)

F. Diagnostic evaluation

1. Chest radiograph
   a. Most useful in excluding other disease processes or complications (pneumonia, pneumothorax, atelectasis, effusions, cancer, CHF): >15% of acutely decompensated COPD patients have treatable findings.
   b. Signs of hyperinflation are common (60% of patients)
      (1) Increased AP diameter
      (2) Increased retrosternal airspace
      (3) Increased parenchymal lucency

(4) Low and flattened diaphragm

(5) Long and narrow cardiac silhouette (unless chronic bronchitis predominates the clinical picture, in which case, an enlarged right ventricle is seen on the lateral film)

c. Bullae may also be seen.

2. Arterial blood gases

a. Provide information regarding the seriousness and acuteness of airway compromise in the COPD patient

b. PaO_2 is generally low.

(1) Hypoxemia is common in these patients and worsens as the disease progresses, as well as with acute exacerbations.

(2) It is due to ventilation-perfusion mismatching and can usually be corrected by increasing the amount of oxygen being inspired.

c. PaO_2

(1) Reflects the adequacy of ventilation

(2) Normocarbia may be present early on, but hypercarbia develops with disease progression and may worsen with acute exacerbations.

(3) A rapid rise in the PaO_2 decreases the pH, whereas with a more gradual rise, the kidneys are able to compensate by retaining bicarbonate, which normalizes the pH. Therefore:

(a) An increased $PaCO_2$, a normal pH, and an increased bicarbonate level suggest chronic CO_2 retention.

(b) An increased $PaCO_2$, a decreased pH, and an increased bicarbonate level point to acute respiratory failure superimposed on chronic respiratory insufficiency.

3. Pulmonary function tests

a. More useful in patients with asthmatic bronchitis than with COPD in evaluating the degree of airway obstruction present and in monitoring the response to treatment.

b. Either the FEV_1 (or the PEFR) may be used; both provide a measurable index of airway obstruction.

c. Because bronchospasm is not the major component of lung dysfunction in COPD patients, the response to treatment is less than that of asthmatics.

d. Lung volume measurements in COPD patients reveal an increase in the amount of dead space and a decrease in the vital capacity; clinical signs develop when the vital capacity drops 50%.

4. ECG

a. Detects ischemic disease or dysrhythmias associated with COPD (especially multifocal atrial tachycardia and atrial fibrillation, both of which are common and should be assumed to be secondary to CHF) and may also demonstrate evidence of COPD or cor pulmonale

(1) P pulmonale (peaked P waves in II, III, and AVF) → very suggestive of COPD

(2) Low voltage, right axis deviation and poor R wave progression → also very suggestive of COPD

(3) Criteria for right ventricular hypertrophy → very suggestive of cor pulmonale

b. Troponin elevation is relatively common in COPD patients with acute exacerbations and is not usually associated with a codiagnosis of acute coronary syndrome.

G. Management

1. Directed toward improving oxygenation and respiratory function
2. Oxygen is the most important therapy.
   a. COPD patients who chronically retain CO_2 depend on their hypoxic respiratory drive to breathe. Raising the pO_2 too quickly in these patients depresses the respiratory center and shuts off this drive. Concern over suppressing this drive must be balanced against the need to increase the pO_2 to a satisfactory level.
   b. To minimize the risk, begin treatment using low concentrations of oxygen and a controlled delivery system such as a Venturi mask. Start with an FiO_2 of 24%–28%, and increase it as needed. The goal is to raise the pO_2 to >60 and the saturation to >88%.
   c. Continuous pulse oximetry (supplemented with arterial blood gases when there is concern that the pCO_2 is rising) should be used to monitor response to treatment and guide adjustments in the FiO_2; continuous capnometry can also be useful.
   d. Assurance of adequate oxygenation should take precedence over concern about suppressing the hypoxic respiratory drive; if a high concentration of oxygen is needed, it should be administered.
3. ß-adrenergic agonists
   a. Produce the most rapid response and should be used for initial therapy; the newer $ß_2$-selective agents are best in that they produce fewer adverse effects.
   b. Inhaled agents: if the patient can provide the necessary inspiratory effort, aerosolized therapy (via a hand-held nebulizer) is preferred; it is associated with equal or superior bronchodilation and less systemic toxicity than parenteral therapy. Delivery via a meter-dosed inhaler with a spacer device may be as effective but requires more patient effort. The usual agents are:
      (1) Albuterol 0.5% solution, 2.5 mg in 2–3 mL normal saline every 20–30 minutes as needed up to 3 doses, then hourly
      (2) Metaproterenol sulfate 5% solution, 15 mg in 2–3 mL normal saline every 20–30 minutes as needed up to 3 doses, then hourly
   c. Subcutaneous medications: reserved for patients who are moving little or no air so that aerosolized therapy is not an option. Available agents include:
      (1) Epinephrine 1:1,000 solution, 0.3 mg every 20–30 minutes as needed up to 2–3 doses
      (2) Terbutaline 1 mg/mL solution, 0.25 mg every 20–30 minutes as needed up to 2–3 doses
      (3) If signs of drug toxicity develop (eg, increasing tachycardia, ventricular ectopy), the dosing interval should be prolonged.
4. Nebulized anticholinergic agents
   a. Should be used in conjunction with ß-agonists as first-line therapy in COPD patients with an acute exacerbation
   b. Unlike the ß-agonists (which have their primary effect on the small peripheral airways), these agents preferentially dilate the larger central airways.
   c. They are as effective as, and in some cases more effective than, ß-agonists in improving pulmonary function; however, they have a slower onset of action (5–15 minutes) and take a longer time to peak (1–2 hours).
   d. These agents are synergistic with ß-agonists and should, therefore, be administered at the same time.

e. Ipratropium bromide is the agent of choice; it has limited systemic absorption and produces far fewer side effects than atropine.
   (1) Dosage: 500 mcg in 2 mL normal saline every 3–4 hours
   (2) Should be added to the nebulizer along with the standard dose of the ß-agonist being administered

5. Methylxanthines (aminophylline, theophylline)
   a. Have a role in the chronic outpatient management of COPD (particularly in patients with nocturnal dyspnea); their role in the treatment of acute exacerbations is currently limited.
   b. Are weak bronchodilators but also function to stimulate the respiratory drive, enhance diaphragmatic contractility, and improve mucociliary clearance.
   c. Can be considered for patients who are not responding well to optimal treatment with ß-adrenergic agonists and ipratropium; studies have demonstrated synergistic activity when all three agents (ß-agonist, anticholinergic, and theophylline) are used.
   d. A serum theophylline level should be obtained immediately in patients who have been taking this medication (8–12 mg/mL is appropriate). The patient may be placed on a maintenance drip until the level is back.
   e. Dosage guidelines for IV aminophylline
      (1) Loading dosage: 5 mg/kg (ideal body weight) over 10–15 minutes
      (2) Maintenance dosage: based on ideal body weight and associated conditions
         (a) Adult smokers <50 years old: 0.9 mg/kg/hr
         (b) Adult nonsmokers <50 years old: 0.5 mg/kg/hr
         (c) Adults >50 years old: 0.4 mg/kg/hr
         (d) Patients with CHF or liver disease: 0.25–0.35 mg/kg/hr
   f. Aminophylline also promotes diuresis and has an inotropic effect. Subsequent potassium depletion may lead to cardiac dysrhythmias.
      (1) Because CO_2 retention causes additional potassium wasting and hypochloremic alkalosis, supplemental KCl should be given if the serum potassium is below normal.
      (2) In addition to the inotropic effects of aminophylline, COPD patients also have a high incidence of cardiac disease. Therefore, cardiac monitoring is indicated during therapy.

6. Steroids
   a. Use in COPD inproves lung function and respiratory symptoms and decreases hospital length of stay
   b. Decrease inflammation and increase responsiveness to ß-agonists and theophylline
   c. The preferred agent for severe exacerbations is methylprednisolone at 80–125 mg IV. Mineralocorticoids (eg, hydrocortisone) should be avoided; they promote sodium retention and potassium excretion.
   d. Patients who receive steroids in the emergency department and are subsequently discharged should be continued on oral prednisone at home. Start with a dosage of 40–60 mg/day for 10–14 days and taper over 1 week or as tolerated.
   e. Patients who are in less severe distress (and will be discharged) may be given oral steroids only; a 2-week tapering regimen of prednisone is recommended.

7. Antibiotics
   a. Should be administered to patients with signs or symptoms of an associated respiratory infection (increase in sputum volume, change in sputum character or color)

b. If signs of pneumonia are present, hospitalization is often required.
c. The most commonly implicated organisms include *Streptococcus pneumoniae, Haemophilus influenzae,* and *Moraxella catarrhalis*.
d. If the patient has acute bronchitis, empiric oral antibiotic therapy aids resolution of COPD exacerbations.

H. Complications

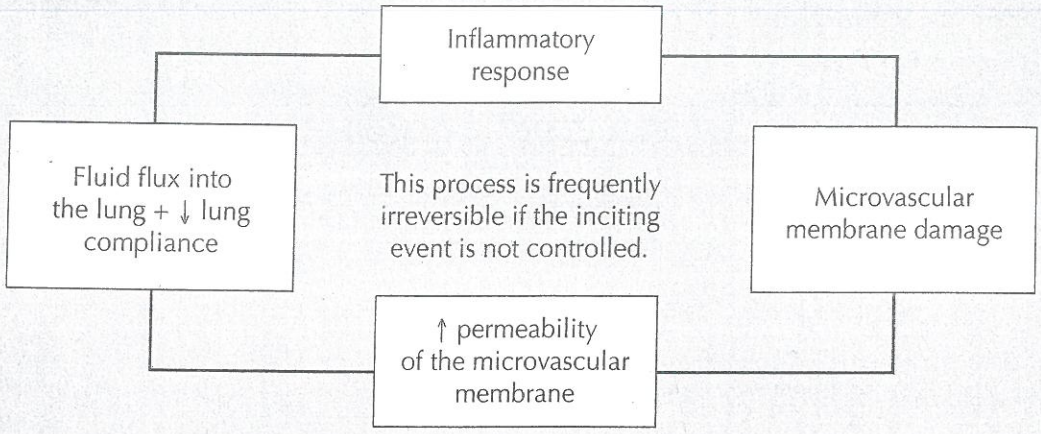
1. Respiratory decompensation → intubation may be needed (beware of ventilator complications, especially tension pneumothorax)
2. Cor pulmonale → phlebotomy may be needed to keep the hematocrit <55%.
3. Multifocal atrial tachycardia
4. Pulmonary embolus
5. Pneumothorax
6. Decreased PO_4 (phosphate)

XI. ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

A. Etiology

1. ARDS is a noncardiogenic pulmonary edema that leads to:
   a. Severe hypoxemia unresponsive to increased concentrations of inspired oxygen (clinical hallmark)
   b. Intrapulmonary shunting
   c. Reduced lung compliance
   d. Potential parenchymal lung damage (pulmonary fibrosis)
2. Sepsis (especially gram-negative sepsis) is the most common cause of ARDS, and aspiration is the second most common cause; together they account for >50% of cases.
3. Precipitating conditions
   a. Severe sepsis
   b. Substance abuse (cocaine, ASA, opiates, tricyclic antidepressants, phenothiazines)
   c. Toxic inhalation (smoke, corrosives, paraquat, oxygen)
   d. Liquid aspiration (gastric contents, fresh- or saltwater)
   e. Pneumonia (bacterial or viral)
   f. Emboli (pulmonary, fat, air, amniotic fluid)
   g. Major trauma (burns, pulmonary contusion, multiple injuries)
   h. Transfusion-related acute lung injury
   i. Pancreatitis
   j. Acute neurologic crisis (head injury, subarachnoid hemorrhage, stroke, brain tumor)
   k. Cancer therapy (tumor lysis, radiation)

B. Pathophysiology



Pathophysiology of ARDS

1. The inflammatory response results from activation of enzyme systems (which is caused by the inciting event) and involves a variety of biochemical mediators that typify an inflammatory response.
2. In many disorders associated with permeability pulmonary edema, the activation of enzyme cascades leads to microvascular membrane damage and increased permeability of the microvascular membrane. There are primarily two enzyme cascades that are activated:
   a. The complement system is triggered by:
      (1) Antigen-antibody complexes (the classic pathway)
      (2) Endotoxins
      (3) Exposure to cell surfaces by bacteria or fungi
      (4) Complex polysaccharides
   b. The coagulation pathways are activated when collagen in exposed basement membrane is exposed to plasma. This causes activation of Hageman factor (factor XII) which, in turn, activates components of the coagulation system, the fibrinolytic system, and the kinin generation system.
3. In addition, metabolism of arachidonic acid through major biochemical pathways leads to the release of a variety of mediators (including a host of leukotrienes and prostaglandins) that contribute to further endothelial and alveolar injury.
4. Neutropenic patients may also develop permeability pulmonary edema. Injury to the pulmonary endothelium results from one of the following:
   a. Direct effects of bacterial endotoxin and complement
   b. Oxygen toxicity
   c. Release of prostaglandins and leukotrienes from alveolar macrophages
5. Once the microvascular membrane is damaged and its permeability is increased, it is no longer an effective barrier to protein flux. Any subsequent increase in hydrostatic pressure then accelerates fluid flux into the lung.

6. The mechanical properties of the lung are also adversely affected: surfactant is reduced or inactivated → collapse of alveoli → stiff lung (which requires greater pressure to inflate it) → ↑ lung compliance

C. Diagnostic evaluation

1. Diagnostic criteria
   a. Symptom onset is within 1 week of the clinical insult.
   b. Chest radiograph: diffuse bilateral interstitial and alveolar infiltrates with a normal-sized heart consistent with pulmonary edema (noncardiogenic pulmonary edema)
   c. Respiratory failure not fully attributed to heart failure or volume overload.
   d. Oxygen impairment as measured by PaO_2/FiO_2 with PEEP setting >5.
      (1) Mild: PaO_2/FiO_2 >200 but <300
      (2) Moderate: PaO_2/FiO_2 >100 but <200
      (3) Severe: PaO_2/FiO_2 <100
2. A radiologic "pearl" that is helpful in distinguishing ARDS from CHF is the following:
   a. Pulmonary edema and a small heart → ARDS
   b. Pulmonary edema and a large heart → CHF may be contributing but does not exclude ARDS

D. Management: primarily supportive

1. Definitive therapy is directed toward the underlying cause.
2. Supportive therapy involves maintenance of acceptable oxygenation and hemodynamic competence.
   a. The patient with moderate to severe edema should be intubated and put on a mechanical ventilator.
      (1) Incremental oxygen supplementation can be tried first but is frequently ineffective in improving pO_2.
      (2) The use of positive-end expiratory pressure (PEEP) can improve oxygenation deficits and should be instituted if administration of supplemental oxygen does not improve pO_2.
         (a) PEEP stabilizes fluid-filled alveoli that are susceptible to collapse and, therefore, increases the number of alveolar units that can participate in gas exchange during ventilation with PEEP. PEEP also allows reduction of FiO_2 to safer levels ($FiO_2 \leq 0.5$).
         (b) As the level of PEEP is increased, observe the patient carefully for adverse effects.
            i. ↑ Venous return → ↓ cardiac output and oxygen delivery
            ii. Pulmonary barotrauma (pneumothorax, pneumomediastinum)
         (c) Optimal PEEP ranges between 0 and 20 mmHg.
   b. Swan-Ganz catheterization is no longer standard practice for diagnosis of ARDS.
   c. IV fluids may be needed to maintain cardiac output and peripheral perfusion, particularly because a decreased venous return may be precipitated by sudden increases of PEEP.
3. Steroids are not effective in either reversing respiratory failure or improving survival, and increase the incidence of subsequent infections; they should not be administered.

E. Outcome

1. Mortality rate ranges from 40% to 70% and may be decreasing even though the incidence of ARDS is increasing because of higher initial survival rates after emergency care.
   a. 15% of patients die of respiratory failure.
   b. Most die of multisystem organ failure.
2. Sepsis is the most common cause of fatal ARDS.

XII. PULMONARY EMBOLISM

A. Definition and pathogenesis

1. Pulmonary embolism is primarily a complication of deep vein thrombosis (DVT), in which a thrombus from one of the deep veins migrates through the right heart and subsequently lodges in and occludes vessels of the pulmonary arterial circulation.
2. Most pulmonary emboli originate from venous thrombi in the lower extremities and pelvis; except for major trauma and postsurgery (gynecologic) patients, lower-extremity venous thrombi almost always start in the calf veins.
3. Other less common sources include hepatic, renal, and ovarian veins, right side of the heart, paradoxical left-to-right cardiac shunts, vena cava, and neck veins (especially central venous catheter sites).
4. <10% of pulmonary emboli cause pulmonary infarction, and most of them occur in patients with left ventricular failure and underlying pulmonary disease (in combination). Because therapy is the same for both entities, clinical distinction is unimportant.

B. Clinical importance

1. Significant morbidity and mortality are associated with pulmonary embolism in the United States, and progressive right ventricular failure is the most common cause of death in these patients (usually secondary to massive embolization).
2. The diagnosis is missed antemortem in up to 70% of cases (>400,000/year); one-third of these patients die, while the other two-thirds are at risk of recurrent pulmonary embolism and development of pulmonary hypertension.
3. A significant percentage of patients with DVT develop pulmonary embolism, and many of these cases go unrecognized. The so-called "clinically silent" calf vein thrombus can be deadly. In fact, clinically significant embolization from calf DVT is not uncommon.

C. Risk factors

1. All known risk factors for pulmonary embolism (and DVT) have their basis in Virchow's triad of venostasis, hypercoagulability, and vessel wall injury or abnormality. (Most common risk factors in patients with proven pulmonary embolism are indicated below with an asterisk.)
   a. Current DVT* (high risk of occult cancer)
   b. Obesity*
   c. Prior DVT or pulmonary embolism*
   d. Immobility (including travel) or prolonged bedrest*
   e. Recent trauma (including burns) or surgery (especially orthopedic)*
   f. Carcinoma* or chemotherapy

g. Autoimmune (eg, systemic lupus erythematosus) and immune (eg, AIDS) disorders
h. MI/CHF/COPD/stroke
i. Estrogen therapy
j. Hypercoagulable settings (use of oral contraceptives, pregnancy, postpartum period)
k. Inherited abnormalities of coagulation or fibrinolysis
l. Indwelling central venous catheter
m. IV drug abuse
n. Polycythemia vera

2. Most patients (≥90%) with thromboembolic disease have at least one of these risk factors, although some (those with cancer, inherited abnormalities of coagulation) may not be apparent at the time of presentation. Furthermore, the risk represented by these factors is additive: the greater the number of risk factors present, the greater the risk of pulmonary embolism. Thus, knowledge of whether or not a patient has risk factors for pulmonary embolism can be used to increase or decrease clinical suspicion of the diagnosis. For example: A patient with at least one common risk factor or two or more other risk factors should be considered to have a moderate to high risk of having a pulmonary embolism.

D. Clinical presentation

1. Presenting signs and symptoms of acute pulmonary embolism are determined by the extent of pulmonary vascular occlusion (massive versus submassive) and the patient's baseline cardiopulmonary status.
2. History
   a. Dyspnea (the most common symptom by far)
   b. Pleuritic chest pain
   c. Apprehension
   d. Cough
   e. Hemoptysis
   f. Sweating
   g. Nonpleuritic chest pain
   h. Syncope (more common with massive than submassive emboli)
3. Physical examination findings
   a. Tachypnea
   b. Rales
   c. Accentuated S_2
   d. Tachycardia (heart rate >100 beats per minute)
   e. Diaphoresis
   f. S_3 or S_4 gallop
   g. Clinically evident thrombophlebitis
   h. Decreased breath sounds
   i. Lower-extremity edema
   j. Cardiac murmur
   k. Cyanosis
   l. Hypotension (more common with massive emboli)

4. Dyspnea, pleuritic chest pain, or tachypnea is present in 95% of patients; if all three of these findings are absent, pulmonary embolism is unlikely (especially if there are no risk factors). The "classic triad" of dyspnea, pleuritic chest pain, and hemoptysis is uncommon (seen in <25% of patients).
5. Predicting pretest probability for pulmonary embolism: the Well's Clinical Criteria

Table 13: Well's Clinical Prediction Rule for PE

Suspected DVT	3.0
Alternative diagnosis is less likely than pulmonary embolism	3.0
Pulse >100 beats per minute	1.5
Immobilization (or surgery) within 4 weeks	1.5
History of previous DVT or pulmonary embolism	1.5
History of hemoptysis	1.0
History of malignancy (treatment within 6 months or palliative)	1.0

Total score interpretation

>6 = high pretest probability (66.7% risk)
2–6 = moderate pretest probability (20.5% risk)
<2 = low pretest probability (3.6% risk)

E. Routine screening tests

1. Most useful in excluding other disease processes (MI, pneumothorax, pneumonia, acute pulmonary edema); also provide data that increase or decrease suspicion of pulmonary embolism but, of themselves, do not confirm or exclude the diagnosis
2. Arterial blood gases
   a. Arterial blood gases are rarely used currently because of the less-than-ideal sensitivity and specificity and because of the presence of alternative superior diagnostic tests.
   b. Usually reveal hypoxemia; a PO_2 <80 mmHg is present in 80% of these patients and is often associated with a mild respiratory alkalosis (↓ PCO_2 and ↑ pH) due to hyperventilation/tachypnea.
   c. The finding of hypoxemia increases the likelihood that a pulmonary embolism is present, but a normal PO_2 does not exclude the diagnosis.
   d. The alveolar-arterial (A-a) gradient is a more sensitive indicator of systemic hypoxemia than the PO_2 alone.
      (1) Formula for calculating the A-a gradient at sea level:

$$\text{A-a gradient} = 150 - (PO_2 + \frac{PCO_2}{0.8})$$

      (2) The normal A-a gradient for a patient increases with age and can be calculated with the following formula:

$$\text{Normal A-a gradient} = \frac{\text{Age}}{4} + 4$$

(3) The A-a gradient is abnormally increased in 95% of patients with pulmonary embolism.

(4) The finding of an increased A-a gradient increases the probability a pulmonary embolism is present, but a normal gradient does not exclude the diagnosis; therefore, it should not be used as a "screening" test for pulmonary embolism.

3. Chest radiograph
   a. Useful in excluding other disease processes and also needed to interpret the ventilation/perfusion scan
   b. Abnormal in >80% of patients with pulmonary embolism, but the findings are neither sensitive nor specific
   c. Suspected disease processes in pulmonary embolism
      (1) Atelectasis or pulmonary parenchymal abnormalities (consolidation or patchy infiltrates)
      (2) Elevated hemidiaphragm
      (3) Pleural effusion
      (4) Hampton's hump: a triangular pleural-based density with a rounded apex that points toward the hilum
      (5) Westermark sign: dilatation of pulmonary vessels proximal to the embolus in association with regional oligemia distally
   d. Most common radiographic abnormalities are atelectasis, pulmonary parenchymal abnormalities, an elevated hemidiaphragm, and small unexplained pleural effusions.
   e. Hampton hump and Westermark sign are rare but, when present, are very suggestive of pulmonary embolism.
   f. The presence of a normal chest radiograph (present in <20% of patients) does not exclude the diagnosis. In fact, in the setting of dyspnea and hypoxia, a normal chest radiograph is very suggestive of pulmonary embolism.
4. ECG
   a. A normal ECG is seen in 9%–26% of patients; abnormalities are nonspecific and may include:
      (1) Transient nonspecific ST–T wave changes (most common finding)
      (2) Sinus tachycardia
      (3) Evidence of right heart strain (high correlation in the presence of pulmonary embolism)
         (a) P pulmonale (peaked P wave in lead II)
         (b) Left- or right-axis deviation
         (c) Atrial fibrillation
         (d) Right bundle-branch block (classic but uncommon finding)
         (e) $S_1Q_3T_3$ (classic but uncommon finding)
         (f) Shift in transition zone to V_5 (R and S waves are equivalent)
         (g) T-wave inversions, especially in the inferior and anteroseptal leads
   b. These findings (if new onset) are suggestive of pulmonary embolism.
   c. A normal ECG is found in up to 15% of patients and does not exclude the diagnosis.
5. D-Dimer
   a. Should be used only as a screening tool in low pretest probability patients; cannot be used to make the diagnosis of pulmonary embolism

b. Value of a negative D-dimer

(1) Can exclude pulmonary embolism in patients with low pretest probability using turbidimetric or ELISA quantitative D-dimer <500 alone or

(2) Cannot exclude pulmonary embolism in patients with moderate or high pretest probability without further study, eg, ventilation-perfusion scan (*ACEP Clinical Policy:* Critical Issues in the Evaluation and Management of Adult Patients Presenting with Suspected Pulmonary Embolism)

F. Specific diagnostic tests

1. Ventilation-perfusion (V/Q) scan

a. Essentially a screening test; occasionally diagnostic for pulmonary embolism (or excludes the diagnosis with near certainty) but is most often nondiagnostic

b. Preferred initial diagnostic test of choice for evaluating patients with suspected pulmonary embolism, but this is changing; at least 70% of V/Q scans do not provide the quality of information needed to make appropriate clinical decisions.

c. A pulmonary embolism typically produces an area that is ventilated but not perfused, referred to as a V/Q mismatch; the larger the V/Q mismatch, the greater the likelihood that a pulmonary embolism is present. Therefore, V/Q scans are classified as normal or near-normal; indeterminate (most common reading); or low, intermediate, or high probability of pulmonary embolism based on the number and size of V/Q mismatches present.

d. The V/Q scan result should be interpreted in the context of clinical suspicion of pulmonary embolism, because it increases the predictive value of this test (especially if there is a level of clinical suspicion of pulmonary embolism before obtaining the V/Q scan results).

e. In patients with low to moderate pretest probability, a normal V/Q scan excludes clinically significant pulmonary embolism 98% of the time. A normal perfusion scan also decreases the probability that an angiographically provable pulmonary embolism is present, at least to the point at which further evaluation is indicated only in the presence of high clinical suspicion.

f. An indeterminate or nondiagnostic (low or intermediate probability) V/Q scan neither confirms nor excludes the diagnosis of pulmonary embolism and should not be taken as a diagnostic end point.

g. A high probability scan in a patient with high pretest clinical probability for pulmonary embolism is considered confirmatory and should result in treatment; no further evaluation is required.

h. Advantages

(1) Relatively noninvasive

(2) Can be performed in critically ill patients

(3) Safer for pregnant patients (in whom, after duplex Doppler, it is considered the diagnostic test of choice; it is less invasive and has less fetal exposure than angiography)

i. Disadvantages

(1) Nondiagnostic most of the time

(2) Less useful in patients with lung disease (or abnormal chest radiograph)

(3) Takes time to perform

(4) Requires isotope injection

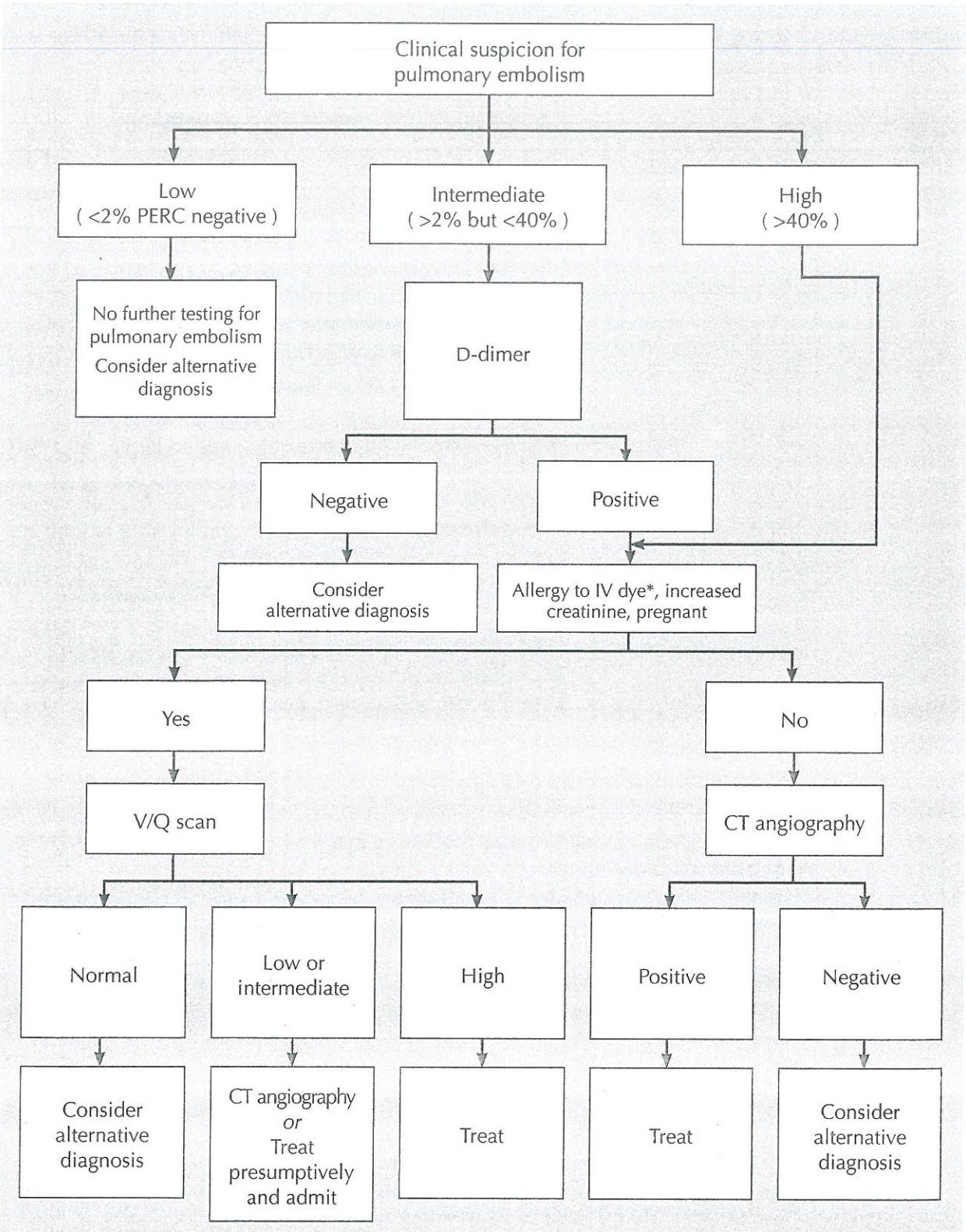
2. CT angiography (CTA)
   a. Most extensively studied new confirmatory test with a good overall test performance that is equal to or slightly better than that of the V/Q scan. However, a CTA scan read as negative for pulmonary embolism does not have as high a negative predictive value as a V/Q scan read as normal; therefore, a negative CTA in a patient with high probability for pulmonary embolism requires pulmonary angiography.
   b. In settings in which a V/Q scan reading is indeterminate (or likely to be so in patients with pulmonary pathology (eg, COPD, pneumonia, etc), a CTA with contrast is the procedure of choice.
   c. An intraluminal filling defect or vascular occlusion characterizes a positive result; the sensitivity and specificity of CTA are higher for pulmonary embolism in the central vessels than in the peripheral vessels (95% sensitivity for segmental or large pulmonary embolism, 75% sensitivity for subsegmental pulmonary embolism). Newer generation CTA (thin collimation spiral CT) may increase diagnostic accuracy.
   d. Advantages over V/Q scanning
      (1) Can be performed within minutes
      (2) Marked reduction of indeterminate readings; useful for its ability to identify a convincing diagnostic alternative to pulmonary embolism
   e. Disadvantages
      (1) Cost
      (2) Need for patient transport
      (3) Use of iodinated contrast material
      (4) Radiation exposure
3. Pulmonary angiography
   a. Gold standard for diagnosing pulmonary embolism; although false-positive and false-negative results do occur occasionally, angiography offers the best available combination of sensitivity and specificity for diagnosing thromboembolic disease.
      (1) Current techniques are unable to detect obstruction in small, subsegmental arterial branches, so many small peripheral emboli may be missed.
      (2) Augmentation techniques (subselective injections, magnification, radiography, balloon occlusion angiography) are able to demonstrate emboli as small as 1 mm in diameter.
      (3) Standard angiography (without augmentation techniques) may completely miss emboli as large as 2.5 mm in diameter.
   b. A positive study demonstrates an embolus obstructing a vessel (a dye "cut-off" sign) or an intraluminal filling defect (in more than one projection).
   c. Advantage: currently the most reliable test available for diagnosing pulmonary embolism
   d. Disadvantages
      (1) Invasive
      (2) Not universally available
      (3) Mortality of 0.1%–0.5% and morbidity of 1%–5%
      (4) Relatively contraindicated in patients with pulmonary hypertension
   e. Complications
      (1) More common in older patients with underlying cardiopulmonary disease
      (2) Include anaphylactoid reactions, dysrhythmias, cardiac arrest, endocardial injury, and perforation

4. Color-flow duplex ultrasonography
   a. In patients with nondiagnostic V/Q scans, this is the next study of choice in excluding pulmonary embolism, because a lower-extremity DVT confirmed by duplex ultrasound is considered de facto evidence that pulmonary embolism also exists.
   b. Demonstration of a DVT in the setting of suspected pulmonary embolism is an indication to start appropriate therapy.
   c. Negative lower-extremity vascular studies cannot eliminate the possibility of pulmonary embolism and, therefore, do not change the clinical management. A single negative lower-extremity ultrasound duplex study should not be used to exclude pulmonary embolism in patients with moderate to high pretest probability and a nondiagnostic V/Q scan. (*ACEP Clinical Policy*).
   d. In pregnant patients, duplex Doppler is the initial diagnostic study of choice for the evaluation of pulmonary embolism, because it is the least invasive. In these patients, duplex Doppler is performed first; if positive, treatment is started, and if negative, then a V/Q or spiral CTA scan is performed.
5. Echocardiography
   a. Should be considered for patients with shock of uncertain cause in whom the diagnosis of pulmonary embolism is suspected but who is not stable enough for other diagnostic tests
   b. Right ventricular dysfunction in the absence of an acute MI or pericardial tamponade supports the diagnosis of right ventricular failure secondary to pulmonary embolism.
   c. Bedside techniques
      (1) Transthoracic echocardiography rarely provides image resolution adequate enough to visualize the inferior vena cava or cardiac chambers, but it can detect right ventricular strain.
      (2) Transesophageal echocardiography has more consistent cardiac imaging, but it is invasive and requires specialty consult and equipment.
6. Point-of-care ultrasound
   a. Useful in unstable patients when comprehensive echocardiography unavailable or delayed
   b. Pseudonormalization of chamber sizes (1:1 ratio); loss of normal right ventricular to left ventricular ratio of 0.6:1.0
   c. McConnell sign: akinesia of mid free wall with normal movement of cardiac apex
   d. Right ventricular thrombus may be seen in 4% of patients with pulmonary embolism.

G. Diagnostic approach

1. The clinical presentation, history, risk factors, and physical examination findings lead to clinical suspicion.
2. Diagnostic evaluation proceeds to include an ECG and chest radiograph, which may indicate another diagnosis or provide additional evidence in support of the diagnosis of pulmonary embolism.
3. If these tests do not reveal another diagnosis (pneumothorax, MI) that explains the patient's signs and symptoms, then the patient's pretest probability for pulmonary embolism should be determined and a screening test performed.
4. Further management decisions should incorporate assessment of the patient's pretest probability of having a pulmonary embolism and the test results (screening and diagnostic).
5. If CTA reveals pulmonary embolism, the patient should be treated.

6. **If CTA is negative and the study was technically adequate, another diagnosis should be considered.**



*Premedication may be necessary in some clinical scenarios to obtain CT pulmonary embolism.

Evaluation of Pulmonary Embolism

H. Management

1. Objectives
   a. Short term: prevent thromboembolic propagation and additional embolic events and eliminate thromboemboli from the pulmonary vasculature
   b. Long term: prevent or minimize recurrence of pulmonary embolism, chronic venous insufficiency, and development of chronic pulmonary hypertension
2. Basic supportive measures
   a. Assess and stabilize the ABCs: establish an IV of normal saline or lactated Ringer's, provide supplemental oxygen, place the patient on a cardiac monitor, and check a rhythm strip.
   b. If patients are hypotensive, any fluid resuscitation should be performed with extreme care. Volume loading can worsen right ventricular function and can actually worsen blood pressure. If blood pressure or clinical status worsens with fluids, immediately switch to vasopressors (eg, norepinephrine, dopamine) for blood pressure support.
3. Anticoagulation with IV unfractionated heparin or low-molecular-weight heparin (LMWH) given SC
   a. The cornerstone of therapy for pulmonary embolism, heparinization should be started as soon as the diagnosis is strongly suspected (unless contraindications exist); do not wait for confirmatory test results.
   b. The decision to use unfractionated heparin or a LMWH is an arbitrary one, because both appear to be equally effective and safe for the initial treatment of submassive pulmonary embolism. The choice is often made by the admitting/consulting cardiologist.
      (1) Because pulmonary embolism is not (generally) treated on an outpatient basis, some cardiologists still prefer IV unfractionated heparin, because the anticoagulation effect can be monitored more accurately and the cost is less than that of an LMWH.
      (2) Others prefer using an LMWH given SC until the patient can be anticoagulated on warfarin. Again, better bioavailability, fewer blood draws for laboratory studies, and no need for a continuous infusion pump are reasons for this choice.
   c. Heparin prevents propagation of the clot and decreases the risk of further embolic events but does not dissolve clots that are already present. It works by binding to (and enhancing) the activity of antithrombin III, a naturally occurring substance that prevents thrombosis by inhibiting activated coagulation factors of the intrinsic and common pathways, particularly thrombin and Factor Xa.
   d. If unfractionated heparin is the agent selected, administer a bolus of 100 units/kg IV (7,000 units/70-kg patient) followed by a continuous infusion of 18 units/kg/hr. Check the partial thromboplastin time (PTT) in 6 hours, and adjust the infusion rate as needed to maintain it at 1.5–2.5 times the control value. This weight-adjusted heparin regimen minimizes the time needed to achieve a therapeutic PTT without increasing the likelihood of bleeding complications.
   e. Enoxaparin is the LMWH of choice and is rapidly becoming the therapeutic method preferred by many clinicians; the dosage is 1 mg/kg SC every 12 hours or 1.5 mg/kg/day SC. Tinzaparin, dalteparin, and nadroparin are acceptable alternatives.
   f. Both unfractionated heparin and LMWHs are safe to use in pregnancy, because neither crosses the placenta. Do not give warfarin in pregnancy!
   g. Complications

(1) Hemorrhage can be reversed by stopping the infusion and administering protamine sulfate (1 mg neutralizes 100 units of heparin but not enoxaparin or other LMWHs).

(2) Thrombocytopenia requires discontinuation of all forms of heparin. It is immune-mediated and generally develops 7–10 days after therapy has started.

h. LMWHs have a number of advantages over unfractionated heparin; when these agents become more widely used for treatment of pulmonary embolism and DVT, they are likely to replace current anticoagulant therapy with unfractionated heparin.

4. Thrombolytic (fibrinolytic) therapy

a. Current indications (*ACEP Clinical Policy:* Critical Issues in the Evaluation and Management of Adult Patients Presenting with Suspected Pulmonary Embolism)

(1) Hemodynamic instability in patients with confirmed pulmonary embolism

(2) Hemodynamic instability in patients with a high clinical index of suspicion (especially with right ventricular dysfunction on bedside echo)

(3) Right ventricular dysfunction on echo in hemodynamically stable patients with confirmed pulmonary embolism

b. Thrombolytic agents work by directly lysing clots.

c. Before administering these agents, the diagnosis of pulmonary embolism should ideally be established with a high degree of certainty. However, if there is a high suspicion of pulmonary embolism in a patient who is hemodynamically compromised, thrombolytic therapy may be administered before diagnostic testing.

d. Administration of these thrombolytic agents should be followed immediately by full-dose heparin anticoagulation (There is no increase in bleeding complications when heparin is administered concurrently with tissue plasminogen activator.)

e. Advantages: in contrast to heparin, thrombolytic therapy prevents the postphlebitic syndrome, reduces the risk of recurrent pulmonary embolism/DVT, restores normal myocardial and valvular function, normalizes pulmonary vascular resistance and pulmonary arterial pressure, improves long-term exercise capacity, and normalizes pulmonary capillary volume and pulmonary gas diffusion. There is also some evidence that it does decrease mortality but this has not yet been clearly demonstrated.

f. Contraindications: identical to those for acute MI (see reperfusion therapy in MI, pages 57–59).

g. Complications

(1) Thrombolytic therapy and anticoagulant therapy produce a similar incidence of systemic bleeding complications. However, the risk of hemorrhage increases directly with the duration of infusion of the thrombolytic agent: urokinase and streptokinase are usually infused over a 12–24 hour period, whereas the usual protocol for TPA is a 2-hour infusion.

(2) If serious bleeding complications occur, the thrombolytic infusion should be discontinued, and aminocaproic acid should be administered along with transfusions of fresh frozen plasma and cryoprecipitate.

h. Thrombolytic agents approved for treatment of pulmonary embolism include streptokinase and TPA; TPA is preferred because it significantly improves hemodynamic parameters in <2 hours (hemodynamic effects are more delayed with streptokinase) and, as indicated above, it is associated with a lower risk of hemorrhage. The dosage of TPA is 100 mg over 2 hours.

5. Surgery
   a. Pulmonary embolectomy
      (1) Rarely performed today; has mostly been supplanted by thrombolytic therapy. More common at tertiary care centers.
      (2) Currently reserved for patients in whom thrombolytic therapy is contraindicated or unsuccessful
      (3) Operative mortality is 25%.
   b. Vena caval filter placement to prevent recurrent pulmonary embolism is indicated in patients with:
      (1) Contraindications to anticoagulation (or major bleeding while receiving anticoagulant therapy)
      (2) Recurrence despite adequate anticoagulation
      (3) Septic emboli arising from the pelvis
      (4) Right-heart failure in those who are not candidates for thrombolysis

XIII. CYANOSIS

A. Definition

1. A bluish discoloration of the skin/mucous membranes that becomes apparent when one of the following is present:
   a. ≥5 g/dL of reduced, unsaturated hemoglobin
   b. 0.5 g/dL of sulfhemoglobin
   c. 1.5 g/L of methemoglobin
2. Cyanosis generally occurs in low oxygen saturation states (such as pO_2 or pulmonary function and cardiac AV shunts).

B. Categories

1. Cyanosis is divided into two categories: central and peripheral.
   a. In central cyanosis, the skin and mucous membranes are affected due to unsaturated arterial blood or the presence of an abnormal hemoglobin derivative.
   b. In contrast, peripheral cyanosis is due to the slowing of blood flow to a specific area of the body (such as the extremities) and an increased extraction of oxygen from normally saturated arterial blood.
2. Etiology of central cyanosis (decreased arterial oxygen saturation)
   a. Decreased atmospheric pressure (high altitude)
   b. Impaired pulmonary function
      (1) Alveolar hypoventilation
      (2) Disparities between pulmonary ventilation and perfusion
      (3) Impaired oxygen diffusion
   c. Anatomic shunts
      (1) Certain types of congenital heart disease
      (2) Pulmonary AV fistulas
      (3) Multiple small intrapulmonary shunts

d. Hemoglobin with low affinity for oxygen
e. Hemoglobin abnormalities (may be drug-induced)
   (1) Methemoglobinemia (acquired and hereditary)
   (2) Sulfhemoglobinemia (acquired)
   (3) Both may result from the use of phenacetin, acetanilid, aniline, and sulfonamides.

3. Etiology of peripheral cyanosis (normally saturated arterial blood with increased oxygen extraction)
   a. Reduced cardiac output (eg, CHF)
   b. Cold exposure (eg, frostbite, severe hypothermia)
   c. Redistribution of blood flow from extremities (eg, shock states)
   d. Arterial or venous obstruction (eg, peripheral vascular disease)

THORACIC AND RESPIRATORY DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow practice clinical scenarios.

Scenario A

Presentation: An elderly male patient presents in respiratory distress with an audible wheeze. He has a significant smoking history, appears malnourished, and has difficulty speaking in complete sentences. He complains of shortness of breath that has been present for several days. The patient has noted a change in the character of his sputum. He does not have a fever.

Physical examination: The patient appears tachypneic, cyanotic, and is showing some use of accessory muscles of respiration. Lung examination reveals grossly diminished air movement throughout with expiratory wheezing. There is no peripheral edema.

What is the diagnosis?

Scenario B

Presentation: A patient presents with symptoms of urinary tract infection/ pyelonephritis. He has not been feeling well, with intermittent flank pain and nausea, for 3 days. There is no history of CHF, liver disease, or malnutrition.

Physical examination: Vital signs are temperature 102°F (39°C), blood pressure 110/64 mmHg, heart rate 130 beats per minute, respiratory rate 22 breaths per minute, and SpO_2 95%. The patient is ill appearing. Chest examination reveals scattered, bilateral rales. The abdomen is soft. There is mild left costoverterbral angle (flank) tenderness. There is no lower extremity edema.

What is the diagnosis?

Scenario C

Presentation: An adult patient presents with chest pain and shortness of breath that began after landing from an 8-hour plane flight. The chest pain is pleuritic in nature.

Physical examination: The patient is no distress. Lung examination is clear. One leg appears swollen compared with the other. Vital signs show tachycardia and tachypnea.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: COPD exacerbation

Management: Begin nebulized albuterol, steroid therapy, and antibiotic coverage for COPD exacerbation. Influenza testing is appropriate when in season. Chest radiograph will show evidence of hyperinflation and flattened diaphragms. Consider venous blood gas testing to evaluate for hypercarbia. Hypoxia will be manifest by increased oxygen requirement. Monitor the patient for improvement or decline in respiratory status. Noninvasive positive-pressure ventilation may be necessary to assist with work of breathing for severe exacerbations. Patients may be discharged if there is immediate clinical improvement in symptoms, and if they appear clinically well and can ambulat safely without evidence of tachypnea or hypoxia. Patients with persistent distress, continued wheezing, or who need frequent nebulized treatments, increased oxygen requirements, or BiPAP should be admitted to an appropriate level of care.

Scenario B

Diagnosis: severe sepsis with evidence of acute lung injury

Management: The patient has acute lung injury from severe sepsis due to pyelonephritis. Chest radiograph may be suspicious for volume overload with bilateral infiltrates; however, this is related to pulmonary capillary leak related to sepsis. The patient is in need of early antibiotic and fluid resuscitation. Noncardiogenic pulmonar edema should not prevent fluid resuscitation. Arterial blood gases can be helpful to determine the degree of acute lung injury. Increased oxygen requirement may require intubation. Intubated patients should be treated with lung protective ventilation of 6 mL/kg ideal body weight based on ARDSNet data.

Scenario C

Diagnosis: pulmonary embolism

Management: There is high clinical suspicion of pulmonary embolism based on history. Empiric anticoagulation may be appropriate for clinically unstable patients. The diagnosis of pulmonary embolism is confirmed by CT study. Hypotensive patients or patients with right ventricular strain may benefit from thrombolysis. Point-of-care ultrasound in the emergency department or comprehensive echocardiography may be helpful. The decision for thrombolysis can be made in conjunction with appropriate consultants (pulmonary, cardiology, radiology, surgery, etc), depending on your institution's practice patterns.

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TRAUMATIC DISORDERS: SELF-ASSESSMENT QUESTIONS

1. The smallest amount of blood loss that consistently produces a decrease in the systolic blood pressure in adults is:

   (a) Loss <15% of blood volume
   (b) Loss 15%–30% of blood volume
   (c) Loss 30%–40% of blood volume
   (d) Loss >40% of blood volume

2. When evaluating a child who has sustained blunt trauma as a result of a motor vehicle collision, which of the following signs would most likely be the earliest and most reliable indication of hemorrhagic shock?

   (a) Altered mental status
   (b) Hypotension
   (c) Tachypnea
   (d) Tachycardia
   (e) Hypoxemia

3. The leading cause of death and disability in trauma victims is:

   (a) Head injury
   (b) Spinal cord injury
   (c) Abdominal injury
   (d) Thoracic injury

4. Which of the following statements regarding cardiac tamponade is inaccurate?

   (a) It is most commonly caused by blunt chest trauma.
   (b) Clinical findings include hypotension, decreased pulse pressure, jugular venous distentio pulsus paradoxus, and muffled heart tones.
   (c) Initial therapy is with IV fluids and pericardiocentesis.
   (d) It can manifest as pulseless electrical activity.

5. A patient with chest trauma is hypotensive on presentation and complains of shortness of breath. Examination reveals jugular venous distention, tracheal deviation, and decreased breath sounds associated with hyperresonance to percussion on one side of the chest. In addition to providing oxygen, starting IV lines, and placing the patient on a cardiac monitor, what is the most appropriate immediate intervention?

   (a) Pericardiocentesis
   (b) Tube thoracostomy
   (c) Intubation
   (d) Needle thoracostomy

6. Of patients with the following acute traumatic injuries, which is most likely to survive at the scene of injury and present to the emergency department with signs of life?

   (a) Complete airway obstruction
   (b) Atlanto-axial dissociation
   (c) Liver laceration
   (d) Aortic rupture
   (e) Cardiac laceration

7. The most common abdominal organ injured in blunt trauma is:

   (a) Spleen
   (b) Liver
   (c) Pancreas
   (d) Kidney

8. All of the following therapies are useful in the treatment of myoglobinuria except:

   (a) Emergency dialysis
   (b) IV mannitol
   (c) Alkalinizing the urine
   (d) Increasing the urine output (by increasing IV fluid resuscitation)

9. All of the following chest radiograph findings are consistent with the diagnosis of traumatic rupture of the aorta except:

   (a) Widening of the superior mediastinum
   (b) Apical pleural cap
   (c) Deviation of the trachea to the right
   (d) Elevation of the left mainstem bronchus

10. Which of the following techniques provides the best assessment of retroperitoneal organs (eg, pancreas, duodenum)?

    (a) Physical examination
    (b) Diagnostic peritoneal lavage
    (c) CT of the abdomen and pelvis
    (d) FAST examination

11. Although all of the following techniques should be used when available, the most reliable method of confirming endotracheal tube position is by:

    (a) Listening over the upper lung fields for equal breath sounds
    (b) Checking the position of the tube on a postintubation chest radiograph
    (c) Noting the presence of condensation in the tube
    (d) Seeing the endotracheal tube pass through the vocal cords

12. Which of the following patients with head injuries is least likely to have a clinically signific traumatic brain injury (ie, an injury resulting in death, neurosurgical intervention, endotrac intubation, or prolonged hospitalization)?

    (a) A 12-month-old who ran into the corner of a coffee table, fell to the ground, and criec immediately, but is now acting normally per his parents; the child has a large frontal hematoma but an otherwise normal examination.

    (b) A 10-year-old who fell 15 feet while climbing a tree and initially had brief loss of consciousness but was awake and alert when EMS arrived; en route to the emergency department, he vomited three times and on arrival he is poorly responsive to voice.

    (c) A 1-month-old who was brought in by her parents after reportedly rolling off the chan table (3–4 feet) and falling to the floor; the parents report that the child cried immedia and has not vomited. On examination, the child is crying and has signs of external tra

    (d) A 75-year-old nursing-home resident who tripped and fell while in the bathroom has a large posterior scalp laceration and hematoma; she is awake and alert, albeit slightly confused, and her neurologic exam is nonfocal.

    (e) A 35-year-old unrestrained driver who was involved in a single car roll-over motor vehicle collision at highway speeds, ejected from the vehicle, and found by EMS in th roadside ditch. He has a Glasgow Coma Score of 13, is confused, and has abrasions c his face, chest, and extremities. The remainder of the primary and secondary surveys i unremarkable.

13. All of the following statements regarding epidural hematomas are accurate except:

    (a) Associated parietal or temporal skull fracture is common.

    (b) CT reveals a lens-like, biconvex lesion.

    (c) Signs and symptoms are due to the mass effect of an arterial bleed.

    (d) Pupillary findings are typically contralateral to the side of the lesion.

14. All of the following statements regarding basilar skull fractures are accurate except:

    (a) Placement of a nasogastric tube and nasotracheal intubation are contraindicated in th presence of these fractures.

    (b) Standard CT scan of the head can reliably exclude basilar skull fractures in patients w clinical suspicion of such injury.

    (c) Clinical findings include CSF rhinorrhea or otorrhea, hemotympanum, raccoon's eyes Battle sign, and the "ring sign" on filter paper.

    (d) Patients should have a neurosurgical consult and possibly be admitted for observation

15. Listed below are several cervical spine injuries and their mechanism of injury. Which is incorrectly matched?

    (a) Jefferson fracture: axial loading mechanism

    (b) Hangman fracture: flexion injury

    (c) Clay-shoveler fracture: flexion injury or direct trauma

    (d) Unilateral facet dislocation: flexion-rotation injury

16. The best view on plain cervical radiographs for visualizing a Jefferson fracture is:

(a) Open-mouth odontoid
(b) Cross-table lateral
(c) AP
(d) Oblique

17. All of the following statements regarding pulmonary contusions are accurate except:

(a) They usually result from blunt chest wall trauma.
(b) Associated rib fractures may be present.
(c) Chest radiograph findings are typically delayed 24–72 hours after injury.
(d) Pneumonia is the most common and most significant complication.

18. Cardiac contusions most commonly involve the:

(a) Left ventricle
(b) Right ventricle
(c) Left atrium
(d) Right atrium

19. The earliest clinical finding in a patient with a compartment syndrome is:

(a) Pain
(b) Paralysis
(c) Palpable tenseness and tenderness of the involved compartment
(d) Pulselessness

20. The most common intracranial bleed associated with closed head trauma is:

(a) Subarachnoid
(b) Subdural
(c) Epidural
(d) Intraparenchymal

21. Epidural hematomas frequently occur in association with skull fractures that traverse the groove of the:

(a) Middle meningeal artery
(b) Middle cerebral artery
(c) Anterior cerebral artery
(d) Anterior meningeal artery

22. An 80-year-old patient presents to the emergency department complaining of bilateral arm weakness after she fell to the ground with walking her dog on a leash. Examination findings reveal 3/5 strength in both arms and 4/5 strength in both legs. Her sensation is normal. The most likely mechanism to cause this specific injury pattern is _____.

    (a) Hyperflexion
    (b) Axial loading
    (c) Rotational forces
    (d) Penetrating injury
    (e) Hyperextension

23. Which of the following treatments used for increased intracranial pressure is most rapidly effective?

    (a) Mannitol
    (b) Furosemide
    (c) Ventilation to a $PaCO_2$ of 30–35 mmHg
    (d) Steroids

24. Which of the following characteristics of a thoracolumbar compression fracture is not associated with vertebral column instability?

    (a) Compression fractures at multiple levels
    (b) Loss of >10% vertebral body height
    (c) Kyphosis >30%
    (d) Rotational component of fracture
    (e) Posterior vertebral wedging

25. Of the following modalities, which is least useful in identifying pneumothoraces?

    (a) Ultrasound
    (b) Upright, expiratory chest radiograph
    (c) CT of the chest
    (d) Supine, inspiratory chest radiograph
    (e) Physical examination

26. Advantages of FAST exam include all of the following except:

    (a) High sensitivity for identifying intra-abdominal hemorrhage
    (b) High specificity for identifying intra-abdominal injuries
    (c) May be performed at the bedside
    (d) May be repeated if clinical condition deteriorates
    (e) Noninvasive

27. All of the following findings are suggestive of a cord injury except:
    (a) Diaphragmatic breathing
    (b) Priapism
    (c) Flaccid areflexia
    (d) Hypertension and bradycardia

28. The presence of which of the following is the most accurate parameter when evaluating diagnostic peritoneal lavage fluid for suspected intra-abdominal injury?
    (a) Bile
    (b) Red blood cells
    (c) White blood cells
    (d) Amylase
    (e) Lactate dehydrogenase

29. Rh immunoglobulin administration would be indicated in all of the following Rh-negative patients except:
    (a) G3P2; 38 weeks gestation; tripped and fell onto abdomen this morning; denies pain, contractions, vaginal bleeding, or discharge
    (b) G6P5; 30 weeks gestation; victim of domestic violence (punched and kicked in face, abdomen, back); complains of painless vaginal spotting
    (c) G1P0; 25 weeks gestation; restrained passenger in low-speed motor vehicle collision; no complaints
    (d) G1P0; 25 weeks gestation; tripped and fell while trail running yesterday, landing on knees and then abdomen; complains of decreased fetal movement but not pain, cramping, bleeding, or discharge
    (e) All of the women should receive Rh immunoglobulin.

30. Which of the following organs is least likely to be injured as a result of primary blast injury?
    (a) Eye
    (b) Ear
    (c) Lungs
    (d) Liver
    (e) Colon

ANSWERS

1. c	6. c	11. d	16. a	21. a	26. b
2. d	7. a	12. a	17. c	22. e	27. d
3. a	8. a	13. d	18. b	23. c	28. b
4. a	9. d	14. b	19. a	24. b	29. e
5. d	10. c	15. b	20. a	25. d	30. d

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. GENERAL INFORMATION

A. Trauma is the leading cause of death in people 1–44 years old worldwide. Many of the injuries are readily treatable (and death is preventable).

B. The primary role of the emergency physician is to assess, resuscitate, and stabilize the trauma patient on a priority basis; this includes both suspicion for and recognition of potentially serious injuries and complications. Trauma management is best provided by a multidisciplinary team, including emergency medicine, general (trauma) surgery, and various surgical subspecialities.

C. Trauma victims have a significantly lower likelihood of morbidity and mortality when treated at a designated trauma center.

D. Trauma-related mortality has a trimodal distribution, ie, three peak times for trauma deaths.

1. Immediate death
   a. Occurs within seconds to minutes of injury
   b. Generally die at the scene
   c. Accounts for greatest number of trauma deaths
   d. Death is usually due to:
      (1) Massive head injury
      (2) High cervical spine injury with high spinal cord disruption
      (3) Cardiac laceration
      (4) Aortic rupture
      (5) Laceration of other great vessels
      (6) Airway obstruction
2. Early death
   a. Occurs within minutes to a few hours of injury, the so-called "golden hour"
   b. Generally die within 4 hours of arrival at trauma center
   c. Death is generally secondary to:
      (1) Subdural and epidural hematomas
      (2) Ruptured spleen or lacerated liver
      (3) Multiple injuries associated with hypovolemic shock
      (4) Fracture of the pelvis or multiple long bones
      (5) Hemopneumothorax
      (6) Tension pneumothorax
      (7) Cardiac tamponade
      (8) Massive hemothorax
      (9) Aortic dissection/rupture
      (10) Airway compromise
   d. Application of advanced trauma life support (ATLS) guidelines regarding rapid ABC assessment and stabilization (definitive airway management, fluid resuscitation, and blood replacement) reduces the mortality and morbidity rate of these patients.

3. Delayed death
   a. Occurs days to weeks after the initial injury
   b. Usually die in intensive care setting
   c. Death is usually due to:
      (1) Multisystem organ failure
      (2) Systemic inflammatory response syndrome
   d. With advances in trauma critical care management, relatively few trauma victims die beyond 24 hours after injury. Therefore, some experts suggest that distribution of trauma mortality is bimodal rather than trimodal.

II. GENERAL APPROACH TO THE TRAUMA PATIENT

A. ATLS guidelines provide a clear, organized, and rapid approach to the evaluation and management of trauma patients.

B. Primary survey: assessment and management of the ABCs
   1. Airway and cervical spine assessment
      a. Airway management is the single most important prehospital/arrival priority.
      b. Immediately assess the airway for potential compromise (nonpatent or poorly protected airway). Check the following:
         (1) Airway patency
            (a) If the patient can speak, the airway is patent.
            (b) If the respirations are noisy (stridulous, sonorous, gurgling, etc), partial obstruction is present.
         (2) Airway protection
            (a) Assume the airway is poorly protected if:
               i. Gag reflex is depressed or absent
               ii. Secretions are pooling
               iii. Decreased level of consciousness (Glasgow Coma Score ≤8)
            (b) The poorly protected airway is at risk of aspiration of gastric/oral secretions, blood, food, foreign bodies.
      c. Beware of the potential for associated cervical spine injuries.
         (1) *Always* maintain the cervical spine in neutral position with inline manual cervical immobilization or appropriate immobilization devices when evaluating and managing the airway.
         (2) Significant cervical spine injury should always be suspected in the patient with:
            (a) Blunt trauma
            (b) Multiple injuries
            (c) Altered level of consciousness
            (d) Evidence of blunt injury above the clavicle
            (e) Dive injuries

d. Airway management in patients with possible cervical spine injury
   (1) Open the airway using the chin lift or jaw thrust maneuver. *Do not* use head tilt maneuver.
   (2) Remove dentures and foreign bodies if present.
   (3) Consider placement of an oropharyngeal (conscious patient) or nasopharyngeal (unconscious patient) airway.
   (4) Patients who do not have a patent airway or are not protecting their airway require definitive airway management (orotracheal intubation or a surgical airway).
   (5) Orotracheal laryngoscopy and intubation with in-line cervical spine immobilization is the procedure of choice (even in the presence of suspected cervical spine injury).
      (a) Front of the cervical immobilization device should be removed while maintaining in-line cervical spine immobilization to facilitate intubation.
      (b) Direct laryngoscopy may be more difficult in the trauma patient.
      (c) Video-assisted laryngoscopy has been shown to have a high success rate of intubation with less movement of the cervical spine.
      (d) Fiberoptic laryngoscopy is an option if the patient's condition permits and the physician is skilled in this technique.
      (e) An endotracheal tube introducer (gum elastic bougie) is a valuable tool in airway management in trauma patients.
   (6) Failed airway management
      (a) If orotracheal intubation is unsuccessful or relatively contraindicated (see below), surgical airway is indicated.
      (b) Surgical cricothyrotomy is surgical airway of choice for the emergency physician. (Some authors consider a surgical airway the procedure of choice for patients with severe midfacial injuries; however, most clinicians would make a single attempt at orotracheal intubation with in-line manual cervical immobilization before proceeding to a surgical airway.) In children <12 years old, needle cricothyrotomy followed by transtracheal jet insufflation is the procedure of choice (because the cricothyroid membrane is not yet palpable).
      (c) Airway alternatives: a laryngeal mask airway or esophageal-tracheal device (Combitube® or King LT Airway®) may be used as a bridging device; neither is considered a definitive airway.
   (7) After successful intubation, verify the position of the endotracheal tube by:
      (a) Listening over lung fields (including apices) for equal breath sounds
      (b) Listening over the epigastrium to exclude the presence of the sound of air in the stomach with positive-pressure ventilation
      (c) Checking the position of the endotracheal tube on a chest radiograph taken after intubation
      (d) End-tidal CO_2 continuous monitoring or colorimetric detection device (When cardiac arrest confounds attempts to assess placement using end-tidal CO_2, an aspiration device, eg, esophageal detector device, may be useful.)
      (e) Although all of the above techniques should be used, the most reliable method of confirming endotracheal tube placement is seeing the tube pass through the vocal cords.

2. Breathing
   a. Observe the rate, depth, and pattern of respiration.
   b. Place the patient on a pulse oximeter to assess the adequacy of oxygenation.
   c. Administer supplemental oxygen to all trauma patients; use high-flow oxygen (10–12 L/min) to deliver an FiO_2 >0.85.
   d. Observe, palpate, and auscultate the thorax to quickly identify life-threatening injuries that may compromise cardiopulmonary function.
      (1) Tension pneumothorax: perform needle thoracostomy, and follow immediately with thoracostomy tube placement.
      (2) Sucking chest wound: cover with a sterile occlusive dressing, taped down on three of its four sides.
      (3) Flail chest: intubation with positive-pressure ventilation indicated for large flail segments.
   e. Ventilator settings in the trauma patient may be different than usual because of changes in hemodynamic status.
      (1) Positive-pressure ventilation in a hypovolemic patient may decrease venous return to the heart (preload) and worsen hypotension; low tidal volumes are appropriate (5 mL/kg at a rate of 20–30 breaths per minute).
      (2) Close monitoring of pulse and blood pressure and frequent arterial blood gases ultimately determine the ventilator settings.
3. Circulation and hemorrhage control
   a. Control obvious external bleeding.
      (1) Direct manual pressure followed by application of a pressure dressing is considered first-line management; elevation of affected extremity may help.
      (2) Application of a tourniquet may be necessary to control life-threatening hemorrhage from a severely injured extremity (eg, explosion injury).
      (3) Bleeding from posterior scalp lacerations are often overlooked initially.
      (4) Various topical hemorrhage control agents have been adapted from those used on modern battlefields and are now commercially available.
   b. Assess circulation for signs of shock.
      (1) Check central and peripheral pulses for quality, rate, and regularity.
      (2) Evaluate skin color, capillary refill time, and level of consciousness.
   c. Take measures to control internal sources of bleeding.
      (1) Unstable pelvic fractures may be temporarily stabilized with a pelvic band to decrease blood loss.
      (2) Reduce and temporarily splint obvious long bone fractures to reduce ongoing hemorrhage.
   d. Place the patient on a cardiac monitor, and check a rhythm strip.
   e. Establish IV access.
      (1) Place two short, large-bore peripheral IV catheters (16-gauge or larger). Draw blood for laboratory studies at the same time.
      (2) If peripheral access is difficult or delayed, place an intraosseous catheter in the seriously injured patient.
      (3) Consider central IV access, but remember that short, large-bore catheters can infuse fluid faster than long central lines.

f. Hemorrhagic shock resuscitation
   (1) Administer 1–2 L warmed lactated Ringer's or normal saline over 5 minutes (fluid bolus in children is 20 mL/kg).
   (2) If the patient remains in shock, packed RBCs should be administered.
   (3) For patients needing large volumes of packed RBC transfusions, consideration should be given to administering other blood products (fresh frozen plasma, platelets) as well.
   (4) There is no standard approach to massive transfusions; protocols vary by institution.
g. Cardiac tamponade
   (1) Classic signs are muffled heart sounds, jugular venous distention, and hypotension (Beck's triad) (may be detected on FAST)
   (2) Perform pericardiocentesis.
h. Indications for thoracotomy in the emergency department (must have surgeon and operating room immediately available for continued resuscitation and definitive repair)
   (1) Penetrating thoracic trauma
      (a) Hemodynamically unstable on arrival to the emergency department despite adequate fluid resuscitation; ideally, the patient loses pulses in front of resuscitation team.
      (b) Pulseless for <15 minutes with signs of myocardial activity
   (2) Blunt trauma—a small subset of patients may benefit; characteristics include:
      (a) Loss of vital signs in the emergency department
      (b) Signs of pericardial tamponade quickly diagnosed on bedside ultrasound
      (c) Cross-clamping the thoracic aorta before exploratory laparotomy may be beneficial in some patients with severe abdominal trauma.

4. Disability (neurologic examination)
   a. Assess pupil size, reactivity, and symmetry.
   b. Determine the patient's level of consciousness; use the AVPU method or the Glasgow Coma Scale (GCS).
      (1) The AVPU method provides a qualitative assessment of the patient's level of consciousness. AVPU is an acronym for:
         Alert
         Responds to **V**ocal (or **V**erbal) stimuli
         Responds to **P**ainful stimuli
         Unresponsive
      (2) The GCS provides a quantitative assessment of the patient's level of consciousness based on the patient's best response in three categories: eye opening, verbal response, and motor response. Scores range from a minimum of 3 (no response) to a maximum of 15 (best response in all categories).
   c. Patients with severe head injury plus an altered level of consciousness (or a GCS ≤8) usually require intubation for airway protection. The presence of nonpurposeful motor responses (4 or fewer) also suggests the need for a definitive airway.
5. Exposure/environmental control: completely undress the patient to facilitate a thorough examination, but remember to cover with warmed blankets to prevent iatrogenic hypothermia.

C. Initial diagnostic testing and continued management

1. Order laboratory studies, eg, type and cross, CBC with platelets, prothrombin time (INR)/partial thromboplastin time, BUN/creatinine, electrolytes, glucose, amylase, lipase, ethanol, toxicology screen, ß-human chorionic gonadotropin, urinalysis, arterial blood gases, creatine kinase, lactate, etc, as appropriate. Selective ordering is recommended.
2. Order the following radiographs unless they are clearly not indicated: cross-table lateral cervical spine, AP chest, and AP pelvis.
3. Perform FAST examination: indicated in all trauma patients with signs of abdominal, pelvic, or thoracic injury; femur fracture; or hemodynamic instability.
4. Provide continuous ECG, pulse oximetry, and blood pressure monitoring.
5. Place nasogastric (or orogastric) tube unless contraindications (eg, cribriform plate fracture, other unstable midface fractures) are present.
6. Insert Foley catheter unless contraindications (eg, blood at the urethral meatus, scrotal hematoma, or a high-riding prostate on rectal examination) are present.
7. Determine if there is a need to transfer the patient to a hospital that can provide a higher level of care. If such a need exists, administrative personnel should begin the transfer process while the secondary survey is completed and the patient is stabilized.
   a. All trauma patients whose injuries exceed the management capabilities of their hospital should be transferred to an appropriate trauma center as quickly as possible (may be mandated in some states).
   b. A complete trauma evaluation is not necessary and should not delay transfer of seriously injured patients.

D. Secondary survey and final case management

1. Obtain an "AMPLE" medical history and inquire about the mechanism of injury.
2. Perform a complete physical examination. Be sure to perform a rectal examination, checking for sphincter tone, blood, high-riding prostate, and bowel-wall integrity.
3. Order additional diagnostic studies as indicated, eg, radiographs of thoracolumbar spine and/or extremities, CT, diagnostic peritoneal lavage, etc.
4. Assess and reassess laboratory data, response to fluids, vital signs, and overall stability of the patient.
5. Administer tetanus booster and antibiotics when indicated.

E. Additional information for pediatric patients

1. **Airway management**
   a. **Consider pretreatment with atropine when performing rapid-sequence intubation.**
   b. **Cuffed or uncuffed endotracheal tubes are now acceptable in children.**
      (1) **Size: (16 + age in years)/4**
      (2) **If using cuffed tube, downsize ½ size.**
   c. **Needle cricothyrotomy is preferred in small children (<12 years old) because the cricothyroid membrane is small and difficult to palpate.**
2. **Shock evaluation**
   a. **The decrease in circulating blood volume required to produce minimal signs of shock is 30%.**
   b. **Normal systolic blood pressure (lowest 5th percentile of normal)**
      (1) **Newborn: >60 mmHg**

(2) 1 month to 1 year: >70 mmHg

(3) >1 year: 70 + 2(age in years)

c. Children have robust compensatory mechanisms and maintain normal blood pressure for a longer time despite presence of significant hemorrhage and shock.

3. Younger children (<11 years old) with cervical spine injuries are most commonly injured in the upper cervical spine (C2–C3).

III. HEMORRHAGIC SHOCK

A. Hemorrhage-induced hypovolemia is the most common cause of shock in the trauma patient.

B. The earliest signs of hemorrhagic shock are tachycardia and cutaneous vasoconstriction (prolonged capillary refill time).

C. Once shock is identified, initial management is volume resuscitation with a warmed crystalloid solution; continued management is determined by the patient's response to initial therapy.

D. The amount of blood loss in an average adult can be estimated based on the patient's initial clinical presentation as follows:

1. Class I hemorrhage: blood volume loss 0–15% (0–750 mL)
   a. Minimal clinical signs and symptoms; slightly increased heart rate (<100 beats per minute), normal blood pressure
   b. Although the healthy adult can easily compensate for this small volume loss via compensatory mechanisms, the trauma patient should be treated with crystalloids; blood products are not required.
2. Class II hemorrhage: blood volume loss 15%–30% (750–1,500 mL)
   a. Clinical signs and symptoms: tachypnea, tachycardia (>100 beats per minute), narrow pulse pressure, delayed capillary refill (>3-4 seconds), usually normotensive, mild anxiety, slight decrease in urine output
   b. Blood products may or may not be required; treat first with crystalloids and monitor response.
3. Class III hemorrhage: blood volume loss 30%–40% (1,500–2,000 mL)
   a. Clinical signs and symptoms: tachypnea, tachycardia (heart rate ≥120 beats per minute), decrease in systolic blood pressure, delayed capillary refill, decreased urine output, change in mental status
   b. Blood products and crystalloids should be used.
4. Class IV hemorrhage: blood volume loss >40% (>2 L)
   a. Clinical signs and symptoms: obvious decompensated shock, tachycardia (heart rate ≥140 beats per minute), decreased systolic blood pressure, extremely narrow pulse pressure, scant urine output, delayed capillary refill, confusion, lethargy
   b. Death is imminent without aggressive management: use blood warmers and administer an initial bolus of 2 L of crystalloid; transfuse type O blood (Rh-negative for premenopausal women; Rh-negative or Rh-positive for men or for women after pregnancy is not an issue). Immediate surgery is often required. The crystalloid to blood replacement ratio is 3:1; the patient should receive 3 mL of crystalloid for each

1 mL of blood lost. Remember that aggressive and continuous volume resuscitation is not a substitute for manual or operative control of hemorrhage. (Consider deliberate hypotension with a mean arterial pressure of 60–70 mmHg until surgical control of hemorrhage is obtained.)

IV. COMMON INJURIES BY ANATOMIC LOCATION

A. Traumatic brain injury (head injury)

1. Overview
   a. Head injury is the leading cause of death and disability in trauma cases.
   b. The most frequent cause of death in patients sustaining severe head injury is uncontrolled intracranial hypertension and subsequent herniation.
   c. Assume a cervical spine injury exists in head-injured patients; if clinical assessment or the mechanism of injury suggests the possibility of neck injury, immobilize the cervical spine.
   d. An altered level of consciousness is the hallmark of brain injury.
   e. Indications for head CT in the injured patient (except for patients with minor head injuries and no symptoms, almost all head-injured adults require CT scanning) and risk factors for abnormal findings on CT include:
      (1) Abnormal level of consciousness or sensorium (GCS <15); this includes intoxicated patients who have a higher incidence of head and other serious injuries than nonintoxicated patients.
      (2) Severe headache
      (3) History of loss of consciousness (unless very brief [<5 seconds]) or persistent amnesia
      (4) Suspected skull fracture
      (5) Any signs of basilar skull fracture (hemotympanum, "raccoon" eyes, Battle sign, CSF rhinorrhea)
      (6) Abnormal neurologic examination, including abnormal pupillary function (especially a dilated, unreactive pupil) and any focal neurologic symptoms (eg, lateralizing extremity weakness or subjective or objective sensory deficits). Patients with pupillary defects or focal motor deficits often have a mass lesion (hematoma) and may need emergent neurosurgery.
      (7) Age >65 years old
      (8) Persistent vomiting
      (9) Dangerous mechanism (eg, pedestrian struck by vehicle, ejection from vehicle, falls from ≥3 feet or ≥5 stairs)
   f. Considerations for head CT in children: The 2009 PECARN Guidelines (Kuppermann, et al) identify children at low risk of clinically significant head injury in whom radiation-related risk of CT imaging may outweigh benefit (high negative predictive value). No CT for:
      (1) <2 years old: normal mental status, no scalp hematoma except frontal, loss of consciousness <5 seconds, nonsevere mechanism of injury, no palpable skull fracture, acting normally per parents

(2) **>2 years old: normal mental status, no loss of consciousness, nonsevere mechanism of injury, no signs of basilar skull fracture, no severe headache**

g. The GCS can be used to categorize the severity of head injury as mild (GCS 14–15), moderate (GCS 9–13), or severe (GCS 3–8); all patients with a GCS <8 (and most patients with a GCS of 8) are comatose.

h. Never assume that brain injury is the cause of hypotension; except as a terminal event, hypotension is seldom the result of isolated head injury.

i. Avoid secondary brain injury by preventing/treating hypoxia, hypotension, ischemia, hypercarbia, hypoglycemia, increased intracranial pressure (ICP), seizures, and hyperpyrexia.

j. The Cushing response to increased ICP consists of hypertension, bradycardia, and a decreased respiratory rate; the hypertension represents the brain's effort to maintain cerebral perfusion pressure. Maintaining the cerebral perfusion pressure (mean arterial pressure minus ICP) at 60–70 mmHg is recommended as a means of improving cerebral blood flow.

2. Specific types of head injuries

a. Diffuse brain injuries (concussions, diffuse axonal injuries): usually do not have mass lesions necessitating emergent neurosurgical intervention

(1) Cerebral concussion

(a) Mechanism: head injury → brief loss of neurologic function

(b) Clinical presentation: clinically variable; may range from minimal to severe and debilitating; rarely associated with any focal findings. Common symptoms include:

i. Brief loss of consciousness (not necessary for diagnosis): results from impairment of the reticular activating system

ii. Antegrade amnesia (with perseveration)

iii. Headache

iv. Nausea and/or vomiting

v. Abnormal sensorium (usually brief)

vi. Difficulty with cognition

(c) Imaging: CT of the head and/or MRI of the brain (if performed) are normal.

(d) Management: cognitive rest and avoidance of repeated head injury while brain is recovering

i. May take 7–14 days or longer

ii. Symptoms lasting >2 weeks may be characterized as postconcussive syndrome

(2) Diffuse axonal injury

(a) Clinical presentation

i. Prolonged coma (often lasting days to weeks) ± posturing

ii. ± Autonomic dysfunction (high fever, hypertension, sweating)

(b) Results in widely scattered microscopic neuronal damage throughout the brain

(c) Imaging: CT of head → no mass lesion; may show diffuse punctate hemorrhages throughout white matter

(d) Management: patients require admission and neurosurgical consult (ICP monitoring often indicated); overall mortality 33%.

(3) Cerebral contusion

(a) Clinical presentation: similar to that of a concussion except:

i. Neurologic dysfunction is more profound and prolonged; patients often demonstrate mental confusion, obtundation, or coma.

ii. Focal deficits may be present if contusions occur in the sensorimotor strip.

(b) Imaging: these lesions are generally found in the frontal and temporal lobes and are visible on CT scan. They may be produced at the site of impact (coup contusions) or at the opposite pole of the brain (contrecoup contusions).

(c) Management

i. Admit these patients for observation and obtain neurosurgical consult. No further therapy is indicated unless complications develop.

ii. Patients with temporal lobe contusions are at significant risk of progression of edema; the resulting mass effect may impinge directly on the brainstem. These patients should be closely observed in a neurosurgical ICU.

(d) Complications: potential delayed complications (hours or days) include development of cerebral edema or an intracerebral hematoma at the site of the contusion, resulting in mass effect (best detected by repeating the CT 12–24 hours after the initial scan).

b. Focal brain lesions (contusions, hemorrhages, hematomas): commonly cause mass effect and necessitate emergency neurosurgery

(1) Epidural hematoma

(a) Mechanism and clinical presentation: head injury causing sudden loss of consciousness (impairment of reticular activating system) as well as intracranial bleeding (usually arterial; most commonly middle meningeal artery) → brain may recover from initial cerebral concussion/contusion, resulting in a lucid interval (≤30%). As bleeding causes increased ICP and mass effect, altered level of sensorium (coma), a fixed and dilated pupil on the side of the lesion, and contralateral hemiparesis develop.

i. Transtentorial herniation compresses cranial nerve III and the corticospinal tract.

ii. Classic presentation is not always present.

(b) Imaging: CT scan demonstrates the classic lens-like, biconvex lesion (be able to recognize on CT). Associated parietal or temporal skull fracture is common with this injury (80%).

(c) Management

i. Immediate neurosurgical consult for surgical decompression

ii. Manage increased ICP (see below).

(d) Mortality is 0–20% depending on neurologic status: normal neurologic examination → 0%; coma → 20% without rapid detection and evacuation

(2) Subdural hematoma

(a) More common than epidural hematomas and more often associated with significant primary, intrinsic brain damage (usually covering the entire surface of the hemisphere)

(b) Risk factors: patients with brain atrophy (alcoholic patients, the elderly) are especially susceptible; brain atrophy → bridging veins spanning a greater distance and being more easily torn

(c) **Mechanism and clinical presentation**

i. **Symptoms develop within 24 hours of injury in patients with acute subdural hematomas but are delayed up to 2 weeks or more, respectively, in patients with subacute and chronic subdural hematomas.**

ii. **Caused by "mass effect" of a venous bleed; symptoms range from a mild headache and confusion to lethargy and coma (with or without localizing signs).**

(d) **Imaging: CT demonstrates the classic crescent-shaped lesion (be able to recognize on CT).**

(e) **Management: same as for epidural hematoma; immediate neurosurgical intervention is usually required (except perhaps for chronic subdural hematomas).**

(f) Mortality rate for acute subdural hematomas ranges from 30% with early evacuation to 60% with later evacuation.

i. Mortality is higher in patients who are unconscious from the time of injury.

ii. Patients with a history of a lucid interval (50%–70%) have a better prognosis.

c. Cerebral edema

(1) Pathophysiology

(a) Head trauma → brain swelling → increased ICP that can lead to brain herniation

(b) Cerebral perfusion pressure = mean arterial pressure minus ICP

(c) Increased ICP can initially be compensated by shifting of CSF and blood from the head into the spinal canal. Once these compensatory mechanisms have been exhausted, however, any additional increase in intracranial volume → a large increase in ICP → decreased cerebral perfusion pressure → brain ischemia and brain cell death

(d) CO_2 is the chief regulator of cerebral blood flow.

i. Increased $PaCO_2$ → dilation of the cerebral vascular bed → ↑ cerebral blood flow → ↑ ICP (CSF pressure >15 mmHg)

ii. Hyperventilation → ↓ CO_2 → constriction of the cerebral vascular bed → ↓ cerebral blood flow → ↓ ICP (Be careful: hyperventilation causes hypoperfusion.)

iii. The most rapid mechanism for lowering ICP is hyperventilation (lowers the $PaCO_2$). However, hyperventilation in the absence of proven intracranial hypertension has been associated with poor neurologic recovery. Until intracranial monitoring is started, it is recommended that patients be ventilated to a $PaCO_2$ of 30–35 mmHg during the immediate postinjury period.

(2) **Signs of increased ICP**

(a) **Headache, nausea, and vomiting**

(b) **Decreasing level of consciousness**

(c) **Cranial nerve VI paresis (one or both eyes adducted)**

(d) **Increased respiratory rate or irregular respiratory pattern**

(e) **Cushing reflex (hypertension and bradycardia): a late (and inconsistent) finding**

(f) **Loss of venous pulsations on funduscopic exam**

(g) Papilledema (may take 1–2 weeks to develop); its absence does not exclude increased ICP and should not delay aggressive treatment.

(h) Progression of neurologic symptoms

(i) Development of signs of herniation

i. Decreased level of consciousness

ii. Posturing

iii. Ipsilateral pupil fixed and dilated, or both pupils are fixed (in midposition).

iv. Loss of oculovestibular reflexes (doll's eyes/ice water calories)

(3) Acute management of ICP

(a) Raise the head of the bed 30°.

(b) Ensure adequate sedation.

(c) Maintain the pCO_2 at ~32–35 mmHg.

i. In the setting of acute herniation, hyperventilation to a pCO_2 of 26–30 mmHg effectively lowers ICP by causing vasoconstriction but should be reserved for life-threatening situations as a bridge to more definitive therapy.

ii. Effects of hyperventilation are short-lived (1–24 hours)

(d) Ensure adequate oxygenation (intubate early in course).

(e) Mannitol

i. Osmotic diuretic that reduces brain volume by drawing fluid out of brain parenchyma and into circulation for excretion by kidneys

ii. 0.25–1 mg/kg IV bolus

iii. Caution should be used in patients with decreased intravascular volume and hypernatremia; also controversial with mass lesions.

(f) Hypertonic saline (3%–23.4%): various dosing regimens (3–5 mL/kg IV bolus)

(g) Consult neurosurgery for consideration of ICP monitoring, seizure prophylaxis, and surgical intervention.

d. Herniation syndromes

(1) The addition of a mass to the closed cranial vault results in increased ICP which, if unchecked, can produce cerebral displacement (herniation).

(2) Uncal herniation: occurs when a unilateral mass pushes the medial aspect of the temporal lobe (uncus) through the tentorial incisura. As the uncus herniates, the following clinical signs develop:

(a) Ipsilateral pupil dilatation and fixation (due to the compression of cranial nerve III)

(b) Increased respiratory rate (or Cheynes-Stokes respiratory pattern)

(c) Coma

(d) Posturing (decorticate [not always seen], then decerebrate)

(e) Apnea and death

(3) Central herniation: occurs when a mass effect compresses the brainstem in a more symmetrical way; this results in a progressive (and diagnostic) loss of brainstem function: ↓ level of consciousness + ↑ respiratory rate or Cheyne-Stokes (thalamic, upper brainstem compression) → fixed midposition pupils (midbrain compression) → posturing, loss of oculovestibular reflexes (pontine compression) → flaccidity, apnea (medullary compression)

(4) Cerebellar tonsillar herniation: downward displacement of the cerebellar tonsils through the foramen magnum, resulting in medullary compression → pinpoint pupils and flaccid quadriplegia (most common motor deficit) → apnea and circulatory collapse

e. Skull fractures

(1) Skull fractures help identify patients at high risk of having or developing an intracranial hematoma.

(2) Imaging: routine skull radiographs are not indicated in the evaluation of patients with traumatic brain injury (*ACEP Clinical Policy:* Neuroimaging and Decision making in Adult Mild Traumatic Brain Injury in the Acute Setting). Significant heac injury requires CT evaluation.

(3) Management

(a) Most patients with skull fractures require neurosurgical consult and admission for observation.

(b) Management is based on type of fracture and presence or absence of underlying brain injury.

(4) Types

(a) Linear, nondepressed skull fractures → require no specific treatment.

(b) Depressed skull fractures → may require operative elevation of the bony fragment if fracture extends below inner table

(c) Open skull fractures → require early operative intervention and prophylactic antibiotics.

(d) "Egg shell" skull fractures → require evaluation for child abuse.

(e) Basilar skull fractures

i. Clinical presentation

- **Periorbital ecchymosis ("raccoon" eyes)**
- **Retroauricular ecchymosis (Battle sign)**
- **Hemotympanum or bloody discharge from the ear**
- **Rhinorrhea or otorrhea**
- **Cranial nerve deficits (I, II, VII, VIII); these signs may not develop for several hours after injury.**

ii. Commonly, the only radiographic sign of a basilar skull fracture may be a blood–air interface in the sphenoid sinus; CT scanning (with thin cuts through the basilar skull) is indicated (but may not demonstrate the fracture).

(5) CSF leaks

(a) May be seen from the ear or nose; associated with intracranial air in 20% of cases

(b) Diagnostic evaluation (testing for CSF rhinorrhea)

i. Place a drop of the fluid on filter paper; a clear halo ring suggests CSF.

ii. Send a sample to the laboratory for glucose determination; if >30 mg/dL, CSF is present. (This test is accurate only if the fluid is clear; bloody fluid may yield a false-positive result).

(c) The major complication is development of a CNS infection.

(d) Most CSF leaks resolve spontaneously with conservative management. However, as many as 10% of patients develop a recurrent post-traumatic CSF leak that increases the risk of delayed bacterial meningitis.

(e) Neurosurgical consult is indicated.

3. Complications of head injuries
   a. Immediate (within 2 days)
      (1) Seizures, traumatic neuronal depolarization, low morbidity
      (2) "Talk and Deteriorate" syndrome
         (a) Most frequent neurologic findings are altered mental status and focal deficits.
         (b) Most frequent cause is epidural hematoma.
   b. Early (usually within 1 week)
      (1) Seizures (may indicate progressive injury)
         (a) Open head injuries (50%)
         (b) Minor head injuries (5%)
      (2) Disseminated intravascular coagulation
      (3) Pulmonary edema (neurogenic)
      (4) Diabetes insipidus (loss of normal antidiuretic hormone secretion)
      (5) CSF leak
      (6) Cardiac dysrhythmias
   c. Late (after 1 week)
      (1) Delayed post-traumatic epilepsy
         (a) Acute intracranial hematomas
         (b) Depressed skull fractures
      (2) Recurrent CSF leak
      (3) Hydrocephalus
      (4) Subdural hydroma
   d. Postconcussion syndrome: symptoms may be noted on initial examination of the patient (or may appear later, eg, 1 week after the injury) and may persist for weeks, months, or years; headache (which may be severe) is the most common complaint, but there may be a host of others (irritability, insomnia, dizziness, anxiety, depression, fatigue, visual complaints, hearing problems, loss of memory, inability to concentrate, and sensitivity to alcohol). There is increasing evidence that these patients actually have organic brain damage. Concussive injury to the labyrinth may result in continued vertigo; exclude perilymph fistula.
   e. Post-traumatic epilepsy
      (1) A high incidence is associated with
         (a) Intracranial hematoma
         (b) Depressed skull fracture
         (c) Seizures occurring within the first week of head injury
      (2) Anticonvulsant therapy
         (a) Fosphenytoin/phenytoin, or phenobarbital: currently the agents of choice in the acute phase
         (b) There is evidence that phenytoin reduces the incidence of seizures within the first week of injury, but not thereafter.

(c) For patients with prolonged seizures, diazepam or lorazepam is used (in addition to fosphenytoin) acutely until the seizure stops.

4. Management
   a. ABCs and immobilize the cervical spine; do a pulse oximetry, analyze a rhythm strip, and determine a glucose level at bedside; all patients with a GCS ≤8 need to be intubated.
   b. Assess the patient's level of consciousness using the GCS. A score of ≤8 is associated with severe head injury; a score of 9–13 is associated with moderate brain injury; a score ≥14 is associated with mild head injury.

Table 14: Glasgow Coma Scale

Eye opening response	Spontaneous	4
	To voice	3
	To pain	2
	None	1
Best verbal response	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Best motor response	Obeys command	6
	Localizes pain	5
	Withdraws (pain)	4
	Flexion (pain)	3
	Extension (pain)	2
	None	1

   c. Assess pupillary function: both equality and response to light. The presence of a sluggishly reactive or fixed and dilated pupil is suggestive of ↑ ICP and uncal herniation.
   d. In patients with a decreased level of consciousness, exclude other potential causes (hypoglycemia, hypoxia, drug overdose, sepsis, etc); administer oxygen, thiamine, D50W (if chemstick low), and naloxone. Avoid administration of glucose to head-injured patients who are not hypoglycemic.
   e. Consider cervical spine injury if any of the following is present:
      (1) Unconsciousness or intoxication
      (2) Cervical pain or tenderness
      (3) Neurologic deficits
      (4). Facial fractures (especially LeFort II or III)
      (5) Mechanism of injury that can result in cervical injury

f. Search for associated injuries; the finding of subhyaloid hemorrhages (preretinal bleeding) in children is suggestive of child abuse (shaking injuries or other trauma) or a bleeding disorder.

g. Obtain CT scan and neurosurgical consult as indicated.

h. If signs of ↑ ICP (eg, dilated pupil) are present:

(1) Elevate head 30°.

(2) Ventilate to a $PaCO_2$ of 35 mmHg.

(3) Obtain CT (immediate) and neurosurgical consult.

i. Frequently reassess the patient's neurologic status (especially level of consciousness) looking for signs of evolution.

B. Facial injuries (extensive facial injuries may necessitate a surgical airway)

1. Midface fractures: the LeFort classification

a. LeFort I: transverse through the maxilla above the roots of the upper teeth

b. LeFort II: extends up into the midface and includes the maxilla, nasal bridge, lacrimal bones, orbital floors, and orbital rims

c. LeFort III (craniofacial dissociation): extends from nasal bridge posteriorly along the medial and inferior orbital walls and laterally through the lateral orbital wall and zygoma; fractures extend back through the nasal bones to the sphenoid base; often associated with CSF leaks

2. Orbital wall fractures: orbital "blowout" fractures most commonly occur through the floor of the orbit but may also occur medially in the region of the lamina papyracea of the ethmoid bone.

a. Orbital floor fractures

(1) Clinical presentation

(a) Pain and diplopia on upward gaze

(b) Enophthalmos

(c) Hypesthesia in the distribution of the infraorbital nerve (ipsilateral cheek and lip)

(d) Limitation of upward gaze due to entrapment of the inferior rectus and inferior oblique muscles

(e) Subcutaneous orbital emphysema

(2) Radiographic findings (CT is imaging modality of choice; however, if plain radiographs are used, fractures are best seen on occipitomental, or "Water" view)

(a) Air–fluid (blood) level in maxillary sinus

(b) Prolapse of orbital tissue into the maxillary antrum ("tear-drop" sign)

(c) Bony disruption of the orbital floor

(d) Clouding of the maxillary sinus

(e) Orbital emphysema

b. Medial wall fractures

(1) Clinical presentation

(a) Epistaxis

(b) Emphysema of the lids or conjunctiva

(c) Limitation of lateral gaze due to entrapment of the medial rectus muscle (uncommon)

(2) Radiographic findings (CT scan)
(a) Unilateral clouding of the ethmoid sinus
(b) Orbital emphysema

c. Management
(1) Exclude associated ocular injuries (eg, rupture of the globe, hyphema), which occur in up to 30% of patients with these fractures; CT scanning may be helpful.
(2) Prescribe decongestants and consider prophylactic antibiotics.
(3) Advise patients to avoid Valsalva maneuvers such as blowing their nose, which ca worsen orbital emphysema.
(4) Refer patients to an ophthalmologist for follow-up; symptoms usually resolve on their own, but surgical correction is sometimes needed. (For further discussion of these fractures, see page 169 in HEENT Disorders.)

3. Nasal fractures
a. Imaging of clinically fractured nasal bones is rarely necessary.
b. If all the following criteria are met, imaging is unlikely to affect urgent management, and patient may follow-up with an appropriate subspecialist in several days.
(1) Tendernesss and swelling isolated to nasal bridge
(2) Can breathe through both nares
(3) No septal deviation
(4) No septal hematoma

4. Zygoma fractures
a. Tripod" fracture: involves zygoma, lateral orbital wall, and maxilla; usually caused by direct blow
b. Displaced fractures usually require surgical repair

5. Mandibular fractures

6. Dental injuries: generally require antibiotics, analgesics, and referral

C. Adult spinal trauma

1. Spinal cord injury

Table 15: Classification of Cervical Spine Injuries by Mechanism of Injury and Stability

Anterior subluxation	Flexion	Potentially unstable
Bilateral facet dislocation	Flexion	Always unstable
Simple wedge fracture	Flexion	Stable
Clay-shoveler's fracture (C6, C7, T1)	Flexion	Stable
Flexion teardrop fracture	Flexion	Extremely unstable
Atlanto-occipital dislocation	Flexion	Unstable
Anterior atlantoaxial dislocation	Flexion	Unstable
Odontoid (C2) fracture	Flexion/varied	Unstable
Unilateral facet dislocation (C1–C2)	Flexion-rotation	Potentially unstable
Rotatory atlantoaxial dislocation	Flexion-rotation	Unstable

Pillar fracture	Extension-compression	Stable
Jefferson fracture (burst of C1)	Axial loading	Extremely unstable
Posterior atlantoaxial dislocation	Extension	Unstable
Hyperextension-dislocation	Extension	Unstable
Extension teardrop fracture	Extension	Unstable in extension
C1 posterior neural arch	Extension	Potentially unstable
Hangman fracture (C2)	Extension	Unstable
Hyperextension-fracture/dislocation	Extension	Unstable

a. Incomplete cord lesions
   (1) **Central cord syndrome**
      (a) **Usually occurs in older patients with spondylosis or congenital stenosis of the spinal canal whose necks are subjected to forced *hyperextension* → vascular ischemia of central cord (spinothalamic and corticospinal tracts) → weakness greater in arms than legs and variable loss of bladder control**
      (b) **Management is generally nonoperative.**
      (c) **Prognosis is excellent.**
   (2) **Anterior cord syndrome**
      (a) **Results from cervical *flexion* injuries → impingement of bony fragments on the spinal canal, cord contusion, or compression of the anterior spinal artery → loss of anterior cord function (complete motor paralysis and loss of pain and temperature sensation distal to the lesion) and preservation of posterior column function (vibration, light touch, position sense)**
      (b) **Immediate neurosurgical consult and emergent magnetic resonance or CT myelography are indicated, because a surgically correctable lesion may be causing cord compression.**
   (3) Brown-Sequard syndrome: usually results from a penetrating injury (gun shot or knife wound) → hemisection of the spinal cord → ipsilateral motor paralysis, loss of proprioception, and vibratory sensation with contralateral loss of pain and temperature sensation
b. Clinical presentation
   (1) Suspect spinal cord injury in:
      (a) Patients with an altered or depressed mental status (including intoxicated patients with head or facial injuries)
      (b) Patients with any focal neurologic deficits
      (c) Significant mechanisms of trauma (especially high-speed motor vehicle collisions, falls, football injuries, and diving accidents)
      (d) Patients with unexplained hypotension and associated paradoxical (relative) bradycardia (spinal shock)
      (e) Elderly patients with suspected minor traumatic injuries
   (2) Examination findings suggestive of cervical cord injury
      (a) Flaccid areflexia

(b) Loss of rectal sphincter tone
(c) Diaphragmatic abdominal breathing
(d) Priapism
(e) Hypotension and relative bradycardia (spinal shock)
(f) Facial reaction to painful stimulus above (but not below) the clavicle

(3) Physical examination
(a) Neurologic examination should focus on identifying focal deficits and attempting to localize the injury to a specific spinal cord level.
i. Sensory deficits should be localized to a specific dermatome if possible (Table 16).
- Occipital pain → C2
- Trapezius discomfort → C5
- The hold-up position (with the hands half closed) or the hands and arms flexed at the chest → C6
- "Burning hands" syndrome (especially in football players) → C6–C7

ii. Testing specific muscle groups may help localize injury (Table 16).
iii. A strong handshake indicates an intact spinal cord down to T1.

Table 16: Spinal Root Muscle and Sensory Distribution

Root	Motor	Sensory
C3	Diaphragm, trapezius	Lower neck to clavicle
C4	Diaphragm	Clavicular area
C5	Biceps, deltoid	Lateral arm at or above the elbow
C6	Biceps	Forearm and radial side of the hand
C7	Triceps	Middle finger
C8	Finger flexors	Lateral aspect of hand
T1	Hand intrinsics	Medial side of forearm
T4		Anterior chest wall at level of nipples
T12		Abdominal wall at level of umbilicus
L2–L3	Hip flexion	
L3–L	Knee extension	
L5		Lateral lower leg and large toe
S1	Ankle plantar flexion	Small toe and sole of foot

(b) Other findings
i. Anterior cord function is assessed by testing motor strength, pain, and temperature sensation.
ii. Posterior cord function is assessed by testing light touch, position, and vibratory sensation.
iii. Anal wink (bulbocavernous reflex): squeezing the penis or clitoris causes contraction of the anal sphincter; this is the last reflex to disappear in

patients with spinal shock or a complete cord lesion and the first to return with improvement.

iv. Sacral sparing: preservation of sensation at the sacrum in a patient with a spinal cord injury (a good prognostic sign)

v. Hypotension associated with bradycardia, warm pink skin, and adequate urine output suggests the presence of neurogenic shock (due to loss of sympathetic tone).

vi. Horner syndrome: unilateral ptosis, miosis, anhydrosis; indicates injury to the cervical sympathetic chain at the level of C7–T2.

c. Diagnostic evaluation

(1) Cervical spine radiographs (note: falling into disfavor; most are using CT)

(a) Cross-table lateral, open-mouth odontoid, and AP views have a high but imperfect diagnostic yield for fractures.

(b) Reliance on the cross-table lateral view alone is unacceptable; 10%–15% of fractures, particularly those involving C7, are not visualized on this view. Also, avoid the error of relying on the lateral view alone before removing the semirigid collar.

(c) Additional views may be needed to visualize C7 and the C7–T1 interspace. A lateral view with axial traction on the arms, a "swimmer's" view, or a transaxillary view may be necessary.

(d) Pseudosubluxation of C2 on C3 (<2 mm) is a normal variant in children.

(e) Most missed fractures are in the C1–C2 area (followed by C7 fractures).

(2) In the awake, alert patient, adequate flexion-extension radiographs (in a patient with a normal 3-view series) may be helpful in evaluating for vertebral column ligamentous instability; rarely useful in acute injuries because associated soft-tissue injuries usually limit the patient's ability to adequately flex and extend neck.

(3) CT scan: initial technique of choice in the evaluation of acute cervical injury in an increasing number of emergency departments because of the following potential limitations of the 3-view cervical-spine series.

(a) Inadequate or "suspicious" radiographs (questionable subluxation, unilateral facet dislocation, or posterior laminar fracture)

(b) "Normal" radiographs in patients with disproportionate pain or a neurologic deficit

(c) Positive radiographic findings requiring further evaluation

i. Fracture or dislocation

ii. Fracture of the posterior arch

iii. Burst fracture with vertebral fragments in the spinal canal

(4) MRI: a useful examination when ligamentous or cord injury is suspected

(5) For the comatose patient with possible cervical spine injury, CT, MRI, and flexion-extension studies under fluoroscopy may be necessary to absolutely exclude a clinically significant injury.

d. Cervical spine "clearance": refers to clinical determination that a trauma patient is at very low risk of cervical spine injury and, therefore, radiographic imaging is not necessary.

(1) Clinical decision rules to identify patients at very low risk of cervical spine injury in whom radiographic imaging is not necessary

(a) NEXUS criteria: no midline cervical tenderness, no focal neurologic deficits, no intoxication, no abnormal sensorium/alertness, no distracting injuries

(b) Canadian cervical spine rules: absence of any high-risk characteristics (age >65 years old, dangerous mechanism, extremity paresthesias) and presence of any low-risk characteristics (simple rear-end motor vehicle collision, ambulatory at scene, upright position in emergency department, no midline cervical tenderness, delayed onset of neck pain)

(2) Patients with negative imaging (plain radiographs or CT scan) but persistent midline tenderness, abnormal sensorium, or abnormal neurologic examination should be maintained in a rigid cervical collar until more advanced imaging (MRI or flexion/extension radiographs) may be performed or until patient can be clinically evaluated by above criteria.

e. Management

(1) Patients with unstable cervical spine injuries should be immobilized with a backboard, rigid cervical collar, side immobilization, and chin and forehead straps anchored to the board.

(2) Advanced airway management (if indicated): options include nasotracheal intubation (if the patient is breathing), careful orotracheal intubation with in-line manual cervical immobilization (video-assisted laryngoscopy may be helpful), and cricothyrotomy (if intubation attempts are unsuccessful).

(3) Complete primary and secondary trauma surveys to identify and manage other injuries.

(4) Obtain immediate neurosurgical consult.

(5) Consider high-dose methylprednisolone in patients with evidence of spinal cord injury.

(a) Controversial: considered a treatment option but not standard of care

(b) Data suggest limited improvement in motor function at cost of increased risk of complications (infection).

(c) Load with 30 mg/kg over 15 minutes, and then infuse 5.4 mg/kg/hr for 23 hours (must be given within 8 hours of onset of deficit).

(6) The need for application of traction with Gardner-Wells tongs should be decided in consultation with the neurosurgeon.

(7) Indications for early surgery

(a) Failure to achieve closed reduction in patients with acute cord injury who have incomplete sensorimotor loss

(b) Failure to maintain reduction

(c) Continued bony encroachment despite reduction

(d) Successful reduction but continued compression demonstrated on myelography

2. Vertebral spine trauma

a. A cervical spine injury should be suspected in any patient with:

(1) An injury above the clavicle

(2) A high-speed vehicular injury

(3) A fall from >10 feet

(4) An electrical injury

(5) An altered level of consciousness

(6) A diving accident

(7) A football injury sustained while tackling

b. Thoracic and lumbar injuries
   (1) Should be suspected in patients sustaining multiple traumatic injuries, particularly if the trauma was to the trunk and the patient complains of pain at these sites or has neurologic findings or complaints.
   (2) In patients undergoing chest and/or abdominal helical CT scanning, screening for thoracic or lumbar spine fractures can be performed on reformatted CT data, obviating the need for plain radiograph screening.
   (3) Chance fractures
      (a) Transverse fractures through the vertebral body caused by flexion about an axis anterior to the vertebral column (most frequently seen after motor vehicle collisions in which the patient was restrained only by a lap belt)
      (b) May be associated with retroperitoneal and abdominal visceral injuries
   (4) Fracture-dislocations
      (a) Caused by extreme flexion or severe blunt trauma to the spine → disruption of the posterior elements (pedicles, facets, laminae) of the vertebra
      (b) Because the thoracic spinal canal is narrow in relation to the spinal cord, fracture-subluxations of the thoracic spine commonly result in complete neurologic deficits.
      (c) Chance fractures and fracture-dislocations are extremely unstable and usually require internal fixation.
   (5) Compression fractures
      (a) Most common thoracolumbar fracture
      (b) Characteristics associated with spinal instability: loss of 50% of vertebral height, compression fractures at multiple levels, rotational component of fracture, and significant kyphosis (>30%)

c. Protect the spinal cord (which ends at L2) until the entire spine has been stabilized.
   (1) Obtain radiographs while the patient is immobilized.
      (a) Patients with neurologic complaints or findings should have imaging even in the absence of bone pain or tenderness in these areas (CT for patients that are unconscious, uncooperative, or have an altered mental status).
      (b) When evaluating the cross-table lateral cervical spine radiograph, be sure to visualize all 7 cervical vertebrae (count them!) and the C7–T1 interspace.
      (c) Do not rely on the cross-table lateral radiograph alone to exclude cervical fractures; open-mouth odontoid and AP views must also be obtained or CT performed.
      (d) Detection of a cervical spine injury necessitates radiographic evaluation of the entire cervical spinal column.
   (2) A CT scan should be ordered if plain radiographs are not clearly negative or if the patient has neurologic complaints or findings associated with "normal" radiographs; for suspicious areas, axial CT scans should be obtained at 3-mm intervals.
   (3) Further evaluation with MRI is indicated if the patient:
      (a) Is obtunded (neurologic examination not possible) or
      (b) Has neurologic complaints or findings with normal plain radiographs or CT
         i. MRI can detect the following injuries:
            • Spinal epidural hematoma

- Herniated disc
- Spinal cord contusion/disruption
- Paraspinal ligamentous/soft-tissue injury

ii. When MRI is not available (or appropriate), CT myelography may exclude the presence of acute spinal cord compression caused by a traumatic herniated disc or epidural hematoma.

(4) Flexion-extension views may be performed in a patient who is awake and cooperative but should be aborted at the first sign of pain.

d. Common bony injuries

(1) Jefferson fracture (C1 ring blowout): an axial loading injury; best seen on the open-mouth odontoid view. Associated C2 fractures are seen in ~40% of these patients.

(2) Odontoid fracture: look for swelling anterior to C2 on the crosstable lateral view and for abnormalities on the open-mouth odontoid view; additional views including tomograms and CT are occasionally needed. There are three types of odontoid fractures:

(a) Type I is rare (involves tip of the dens).

(b) Type II traverses the dens at the junction of the body of C2 (worst prognosis).

(c) Type III involves the vertebral body of C2.

(3) Hangman fracture (unstable bipeduncular fracture of C2): an extension injury

(4) Facet dislocations: may be either unilateral or bilateral; unilateral dislocations result from flexion-rotation injuries, while bilateral dislocations are produced by flexion injuries. The lateral view reveals ≥25% (unilateral) to ≥50% (bilateral) anterior displacement of the superior vertebral body relative to the adjoining inferior vertebral body.

(5) Clay-shoveler's fracture: an avulsion fracture of the spinous process of C6–T3 (C7 most common); results from a flexion injury or a direct blow to the spinous process

(6) Flexion teardrop fracture: associated with significant disruption of posterior ligaments and anterior cord syndrome

e. Immediate neurosurgical consult is required for patients with positive radiographs as well as those with neurologic complaints or findings, ie, sensory or motor.

f. Consider administration of high-dose methylprednisolone (30 mg/kg IV bolus followed by an infusion at 5.4 mg/kg/hr) after neurosurgical consult to patients with evidence of a nonpenetrating spinal cord injury who presented within 8 hours of injury; currently not standard of care.

D. Pediatric spine and spinal cord injuries

1. Epidemiology

a. The pediatric age group accounts for a small percentage of all spinal cord injuries.

b. Etiology of cervical spine fractures/dislocations: motor vehicle collisions (~40%), diving/falling accidents (~35%), and sports injuries (~25%).

c. Children <8 years old have a high incidence of upper cervical spine and craniovertebral junction injuries. Factors that favor this injury pattern are flat (horizontally oriented) facet joints, ligament laxity, disproportionately large head, and relatively weak neck muscles.

d. Children >12 years old have lower cervical spine injuries (similar to adults).

2. Prehospital management

a. Pediatric long-spine board: in children <8 years old, padding should be placed underneath the shoulders to maintain neutral alignment of the spine.
b. Hard cervical collar
c. Securing straps
d. Soft spacing devices (eg, blanket rolls) should be placed between head and securing straps to prevent lateral movement.

3. Radiologic variables
   a. Absent lordosis (~15% of patients) on lateral radiograph
   b. Pseudosubluxation (subluxation up to 2–3 mm can be considered normal)
      (1) Most pronounced at C2–C3 level; 40% of children <7 years old and 20% of children up to 16 years old exhibit anterior displacement of C2 on C3.
      (2) May also be seen at the C3–C4 level
   c. Predens space
      (1) About 20% of children <8 years old have a gap >3 mm.
      (2) Consider all distances >5 mm as abnormal.
   d. Prevertebral soft tissues
      (1) Suggested normal values are less than one-half the width of the adjacent vertebral body at C2 and less than the full width of the adjacent vertebral boday at C6.
      (2) Crying, forced expiration, and neck flexion falsely increase the distance on imaging.
   e. Anterior wedging of cervical bodies (especially C3 and C4)
   f. Spinal cord injury without radiographic abnormality (SCIWORA)
      (1) Incidence: a very rare diagnosis in pediatrics with the increased use of MRI
      (2) With a history of transient paresthesias, assume a potential injury.
      (3) SCIWORA is a diagnosis of exclusion; bony, ligamentous, and disc injuries must be excluded.

E. Thoracic trauma
1. Epidemiology
   a. Thoracic injuries are responsible for 25% of all trauma deaths.
   b. 10%–30% of thoracic injuries require surgical intervention.
2. Pulmonary injuries
   a. Pneumothoraces
      (1) Tension pneumothorax
         (a) This diagnosis should be made clinically (rather than by radiograph), because it can be rapidly fatal because of severe ventilatory and circulatory compromise.
         (b) Clinical presentation
            i. Respiratory distress (late sign = cyanosis)
            ii. Hypotension (caused by decreased preload)
            iii. Tachycardia (late sign = pulseless electrical activity)
            iv. Tracheal deviation (away from the side of the pneumothorax)
            v. Distended neck veins
            vi. Ipsilateral decreased or absent breath sounds

vii. Ipsilateral hyperresonance on percussion

viii. Asymmetrical chest rise (affected side remains hyperinflated)

(c) Management

i. Immediate needle thoracostomy of the affected hemithorax followed by tube thoracostomy

ii. Needle thoracotomy: insert large-bore IV catheter perpendicular to the chest wall in second intracostal space along midclavicular line

(2) Simple pneumothorax

(a) Clinical presentation

i. Decreased breath sounds and hyperresonance to percussion on the affected side

ii. Dyspnea, pleuritic chest pain

(b) Imaging

i. Expiratory, upright chest radiograph is more sensitive than standard inspiratory view.

ii. Ultrasound may be used to detect pneumothorax quickly at bedside.

(c) Management: tube thoracostomy

(3) Open pneumothorax ("sucking chest wound")

(a) If the opening in the chest wall is approximately two-thirds of the diameter of the trachea or more, air moves peferentially through the chest wall defect → ineffective ventilation → hypoxia.

(b) Management

i. Cover the defect with a sterile occlusive dressing (petrolatum gauze, plastic wrap); tape it down on three of its four edges.

ii. Place a chest tube in an area distant from the wound site.

b. Pulmonary contusion (most common potentially lethal chest injury)

(1) Mechanism of injury: usually results from direct chest wall trauma (eg, motor vehicle collision with rapid deceleration)

(2) Clinical presentation includes dyspnea, tachypnea, tachycardia, and chest wall tenderness/ecchymosis at the site of injury.

(3) Associated rib fractures may also be present (less commonly in children).

(4) Arterial blood gases reveal hypoxemia and a widening alveolar-arterial oxygen gradient.

(5) Chest radiograph findings are localized to the site of injury and range from patchy irregular alveolar infiltrates to frank consolidation. Radiograph findings may be delayed up to 6 hours.

(6) Management

(a) The goal is adequate ventilation/oxygenation and perfusion to promote healing and prevent complications (eg, pneumonia, pneumothorax).

(b) Mild to moderate contusions (<18% total lung volume or about one lobe) are treated with supplemental oxygen, pulmonary hygiene (nasotracheal suctioning, chest physiotherapy), and pain control (intercostal nerve blocks).

(c) Severe contusions (>28% total lung volume or more than one lobe) associated with significant hypoxemia (pO_2 <65 mmHg) require early intubation and mechanical ventilation.

i. Use of intermittent mandatory ventilation with lower tidal volumes (5–7 mL/kg) (reduce the risk of barotrauma) and positive end-expiratory pressure to prevent alveolar collapse usually provide much better ventilation/perfusion matching than standard controlled ventilation.

ii. When only one lung has been severely contused and significant hypoxemia persists despite the use of intermittent mandatory ventilation and positive end-expiratory pressure, mechanical ventilation may improve if each lung is ventilated separately with a double-lumen endobronchial catheter; this technique helps prevent hyperexpansion of the normal lung and gradual collapse of the injured, poorly compliant lung. Place the normal lung "down" by turning the patient to the decubitus position (helps improve ventilation/perfusion matching of the contused lung).

(7) Pneumonia is the most common and most significant complication; it significantly worsens the prognosis.

c. Hemothorax

(1) Primary radiographic finding (requires at least 200 mL of fluid to be seen) is blunting of the costophrenic angles on an upright chest film; effusions can easily be missed on a supine film.

(2) 25% of cases have concomitant pneumothoraces; 73% have associated extrathoracic injuries.

(3) Clinical presentation: diminished breath sounds, dullness to percussion, and decreased tactile fremitus (hypotension)

(4) Management

(a) Most hemothoraces are self-limited and need only tube thoracostomy.

(b) Consider autotransfusion in patients with massive hemothoraces.

(c) Thoracotomy is indicated for:

i. Blood loss ≥1,500 mL in the initial chest tube drainage

ii. Persistent bleeding requiring continuous blood transfusion

iii. Patients who remain hypotensive or decompensate

iv. Blood loss >200 mL/hr for 2–4 hours

v. ≥50% hemothorax

3. Blunt cardiac injuries usually result from high-speed vehicular accidents in which the chest wall strikes the steering wheel; they are all associated with potentially fatal complications (arrhythmias, wall motion abnormalities, anatomical ruptures, cardiogenic shock), although they should be viewed clinically as a continuous spectrum of myocardial damage.

a. General principles

(1) Cardiac enzymes have limited role in evaluation or management of blunt cardiac injury.

(2) ECG is neither sensitive nor specific; however, presence of abnormalities on the initial ECG correlates with the risk of developing subsequent cardiac complications.

(a) Findings are variable and may include sinus tachycardia (most sensitive, least specific), premature ventricular contractions, atrial fibrillation, bundle-branch blocks (right bundle-branch block more common), ST–T wave changes.

(b) FAST may reveal pericardial hemorrhage and cardiac tamponade.

(c) 2-D echocardiographic findings: impaired regional systolic function (particularly right ventricular free wall dyskinesis), increased echogenicity ("brightness"), increased end-diastolic wall thickness

(3) General management

(a) Patients with conduction abnormalities (or other significant injuries) should be admitted to a critical care unit.

(b) Asymptomatic patients with normal vital signs, a low-risk profile, and normal ECG may be released after a short period of observation.

(c) Symptomatic patients with a low-risk profile and normal serial ECGs can probably be sent home after 6–12 hours of observation; if there is some concern after this time, consider ordering 2-D echocardiography.

(d) Symptomatic patients with a moderate-risk profile and normal serial ECGs should have echocardiography performed; they may be sent home after a 12-hour observation period if the echocardiogram is clearly normal.

(e) High-risk patients (or those with questionable findings on diagnostic evaluation) should be admitted for observation and cardiac monitoring, because they are at risk of developing dysrhythmias and cardiac dysfunction.

b. Concussion (no permanent cell damage): commotio cordis

(1) A sharp, direct blow to the mid-anterior chest wall "stuns" the myocardium and results in a dysrhythmia.

(2) If the episode resolves (spontaneously or with treatment), there are no histopathologic changes. However, prolonged cellular dysfunction may lead to a nonperfusing rhythm (ventricular fibrillation, asystole) and irreversible cardiac arrest.

c. Contusion (permanent cell injury)

(1) Mechanism of injury: high-speed deceleration → heart moves forward and forcibly strikes the sternum; injuries that occur during early systole or late diastole, when the ventricles are blood-filled, are the most damaging.

(2) Site of injury: right ventricle (most common)

(3) Suspecting this diagnosis is more important than making it, because it is often elusive.

d. Infarction (cell death)

(1) Rare complication of blunt trauma

(2) Usually results from coronary artery occlusion by arterial spasm, intimal tear, laceration, dissection, thrombosis, or compression from adjacent hemorrhage, and edema (which may occur with capillary bleeding in a contused heart)

(3) Patients with preexisting coronary artery disease are at greatest risk.

(4) Diagnosis: ischemic changes on ECG; increased cardiac enzymes (may be delayed)

(5) Management: admission for cardiac monitoring and medical management of myocardial infarction

e. Tamponade (bleeding into the pericardium under pressure)

(1) Penetrating trauma is the most frequent cause, but tamponade can also occur with blunt trauma and iatrogenic trauma (central venous pressure catheter placement, pacemaker insertion); the most common site of perforation from catheter placement is the right atrium.

(2) **Mechanism of injury in blunt injury: rapid deceleration with cardiac compression during early systole or late diastole → blood-filled ventricles → rigid myocardial wall → rents/tears/lacerations → impending myocardial rupture**

(3) **Clinical presentation**

(a) **Beck's triad: hypotension, jugular venous distention, and muffled heart tones; however, jugular venous distention may not be present if there is marked hypovolemia (rarely appreciated clinically).**

(b) **Decreased pulse pressure**

(c) **Rising central venous pressure (earliest response)**

(d) **Kussmaul sign (distention of neck veins with inspiration)**

(e) **Pulsus paradoxus**

(f) **A central venous pressure >15 cm H_2O in association with hypotension and tachycardia (most reliable sign)**

(4) **Diagnostic evaluation**

(a) **Electrical alternans (pathognomonic for tamponade but is rarely present in the acute scenario)**

(b) **Pulseless electrical activity can be a manifestation of cardiac tamponade but also occurs with tension pneumothorax, hypovolemia, etc.**

(c) **The presence of pericardial fluid on ultrasound is highly suggestive of active bleeding in the pericardium.**

(5) **Management: IV fluids followed by pericardiocentesis; definitive treatment is open thoracotomy by a qualified surgeon.**

f. Myocardial rupture (exsanguination)

(1) Usually die within seconds to minutes of injury at the scene

(2) Mechanism of injury: compression of a blood-filled chamber (usually a ventricle but may be an atrium, especially the right); most commonly results from blunt thoracic injury

(3) The patient is protected from immediate exsanguination if the pericardium is intact.

(4) Clinical presentation: usually one of pericardial tamponade in a patient with other known or suspected thoracic injuries (eg, hemothorax, pneumothorax, aortic dissection/rupture)

(5) Diagnostic evaluation

(a) Chest radiograph is almost always abnormal (nonspecific).

(b) Bedside FAST reveals pericardial effusion/tamponade physiology.

(6) Management: fluid resuscitation, immediate decompression of cardiac tamponade (pericardiocentesis)

(a) Pericardiocentesis is only a temporary measure until surgical correction can be undertaken.

(b) If the patient's clinical status deteriorates in the emergency department, emergency thoracotomy and pericardiotomy may be required before definitive repair in the operating room.

4. Traumatic aortic rupture

a. Mechanism of injury: sudden deceleration (≥45 mph motor vehicle collision impact, damaged steering wheel or 20–30 foot fall)

b. Site of injury: most ruptures occur at the ligamentum arteriosum (the point of greatest aortic fixation) just distal to the left subclavian artery and progress from the intima outward toward the adventitia.

c. 80%–90% of patients die at the scene, and as many as 50% of remaining survivors die within 24 hours if not diagnosed and treated expeditiously.

d. Clinical presentation

(1) Retrosternal or interscapular pain (25%): most common

(2) Dyspnea

(3) Harsh systolic murmur over the precordium or in the interscapular area

(4) Upper extremity hypertension in association with decreased or absent femoral pulses (pseudocoarctation syndrome)

(5) Pulse deficits

(6) Voice change or hoarseness (in the absence of laryngeal injury)

(7) Ischemic pain of the extremities

(8) Paraplegia

e. Diagnostic evaluation

(1) Diagnosis is usually suspected from the history of a deceleration injury plus findings on chest radiograph (any, all, or none may be present):

(a) Widening of the superior mediastinum (>8 cm) on an upright PA chest radiograph taken at a distance of 6 feet (most common finding)

(b) Obliterated or indistinct aortic knob (most reliable finding)

(c) Deviation of the trachea and/or esophagus (nasogastric tube) to the right

(d) Depression of the left mainstem bronchus >40° below horizontal

(e) Obliteration of the space between the pulmonary artery and the aorta

(f) Left apical pleural cap (obliteration of the medial aspect of the left upper lobe apex)

(g) Multiple rib fractures

(h) Widening and/or displacement of the paratracheal stripe to the right

(i) Widening of the left or right paraspinous stripe

(j) Left hemothorax

(k) Fractures of the first or second ribs or the scapula

(2) Diagnosis is confirmed by:

(a) Angiography

(b) Contrast-enhanced dynamic spiral CT is extremely accurate for demonstration or exclusion of direct aortic injury.

i. Axial CT images can be supplemented by 2-D multiplanar reformations or 3-D surface contour views that can simulate thoracic aortography → better delineates extent of injury and its relationship to branch vessels.

ii. Spiral CT is very useful, because it permits reconstruction of axial images that allows production of better 2-D multiplanar reformations and 3-D images.

iii. If CT demonstrates traumatic aortic injury (or is not clearly negative), aortography should be performed.

(c) Transesophageal echocardiography is a diagnostic alternative if both angiographic and CT findings are equivocal; however, results are highly operator-dependent and patient cooperation is required.

f. Management

(1) Prompt surgical repair

(2) While awaiting surgery, the patient's blood pressure should be carefully controlled so as to minimize the shearing effect on the intact adventitia of the aorta; IV ß-blockers (eg, esmolol [much easier to titrate than propranolol]) in combination with nitroprusside should be used as needed to maintain a systolic blood pressure between 100 and 120 mmHg.

F. Abdominal injuries

1. Clinical caveats

a. All patients involved in high-speed or bicycle accidents should be evaluated for abdominal injury.

b. Initial examination is benign in 20% of patients with abdominal injury; serial examinations are important.

c. The goal is to identify the presence of a surgical abdomen; prolonged evaluation to determine the specific injury is unnecessary and can be detrimental.

2. Ultrasonography/FAST examination

a. The initial (and sometimes only) diagnostic modality of choice for evaluating both hemodynamically stable and unstable patients with blunt thoracoabdominal trauma

Table 17: Comparison of Diagnostic Peritoneal Lavage, Ultrasound, and CT Scan

Diagnostic Study	Indication	Advantages	Disadvantages
Diagnostic peritoneal lavage	Identify intra-abdominal bleeding and/or signs of bowel perforation in hemodynamically unstable patients	Early diagnosis Very sensitive Detects bowel injury (98% accurate)	Invasive Does not detect injuries to diaphragm and retroperitoneum Less specific than ultrasound or CT May overdiagnose injuries
Ultrasound/ FAST	Identify free intra-abdominal, pelvic, pericardial fluid in hemodynamically unstable or stable patients	Early diagnosis Noninvasive Easily repeatable 86%–97% accurate Performed rapidly	Operator dependent Does not detect diaphragm, bowel, and some pancreatic injuries Bowel gas and subcutaneous air distortion
CT scan	Identify solid or viscus organ injury in hemodynamically stable patients	Most specific for injury (92%–98% accurate)	Cost and time Less sensitive for diaphragm, bowel, and some pancreatic injuries than for solid-organ injuries Usually requires transporting patient out of emergency department

b. Should be used in combination with other diagnostic modalities and clinical assessment
   (1) Unstable patient + positive ultrasound → laparotomy
   (2) Stable patient + positive ultrasound → CT (to exclude the need for operative intervention)
   (3) Unstable patient + negative ultrasound → repeat ultrasound or perform diagnostic peritoneal lavage
   (4) Stable patient + negative ultrasound → observation or CT
   (5) A negative or equivocal ultrasound does not exclude the need for further testing.

c. Advantages
   (1) Noninvasive
   (2) Detects the presence of intra-abdominal, pericardial, or pelvic fluid (extended FAST also detects pleural fluid)
   (3) Rapid, safe, and portable (can be performed in the controlled environment of the trauma suite) and may be repeated as needed based on protocol or change in the patient's condition
   (4) Can be performed without interfering with resuscitative efforts
   (5) Does not require administration of contrast agents
   (6) Has a sensitivity of 60%–95% and a specificity of nearly 100% (serial examinations increase sensitivity)

d. Disadvantages
   (1) Can miss bowel and retroperitoneal injuries
   (2) Cannot differentiate fluids, eg, ascites versus blood
   (3) Presence of >200 mL fluid in intra-abdominal cavity usually necessary for positive examination.
   (4) Imaging is impaired in patients who are markedly obese and in those with subcutaneous air/emphysema or excessive bowel gas.
   (5) Evaluations are operator-dependent, ie, determined by skill level.
   (6) Currently, studies evaluating the efficacy of ultrasound for penetrating trauma are limited and inconclusive.

3. CT

a. The diagnostic study of choice for evaluating hemodynamically stable patients with blunt thoracoabdominal, genitourinary trauma, and/or suspected retroperitoneal injuries; often used in concert with the FAST examination (ie, initial evaluation with the FAST examination followed by CT)
   (1) Normal vital signs + normal ultrasound → CT may be deferred
   (2) Normal vital signs + positive ultrasound → CT may delineate extent of injuries

b. Advantages
   (1) Noninvasive
   (2) Provides information on specific organ injury; when liver or spleen injury is suspected, CT may reliably exclude injuries that require emergent operative intervention (*ACEP Clinical Policy*).
   (3) Can diagnose retroperitoneal and pelvic organ injuries

c. Disadvantages
   (1) Usually necessitates administration of IV and sometimes oral contrast
   (2) Takes more time than diagnostic peritoneal lavage to perform
   (3) Requires an expert to interpret it
   (4) Has a rather high false-negative rate (2%–25%); misses certain injuries, eg, diaphragm, pancreas, bladder, and bowel (the value of CT in assessing patients with penetrating abdominal trauma is limited but growing)

4. Diagnostic peritoneal lavage
   a. Used to identify intra-abdominal bleeding (or bowel injury) that requires immediate laparotomy in the unstable patient if a FAST examination is not available or is inconclusive; it is rapidly performed, readily available, and has a sensitivity of 98%.
   b. Indications
      (1) In blunt abdominal trauma, this includes:
         (a) Suspected or known blunt abdominal trauma in a patient with an unreliable or equivocal examination (due to intoxication with drugs or alcohol, distracting injuries, spinal cord lesion, etc) or injury to adjacent structures (lower ribs, pelvis, or lumbar spine)
         (b) Lap belt sign with suspicion of bowel injury
         (c) Unexplained hypo- or hypertension
         (d) Patient unavailable for serial examinations (ie, requires anesthesia for management of other injuries)
      (2) In penetrating trauma, this includes:
         (a) Stab wounds with known or suspected peritoneal violation
         (b) Gunshot wounds when peritoneal violation is unclear
   c. Contraindications
      (1) The only absolute contraindication to diagnostic peritoneal lavage is an indication for laparotomy (eg, free air under the diaphragm, intraperitoneal bladder rupture).
      (2) Relative contraindications
         (a) Previous abdominal surgery
         (b) Morbid obesity
         (c) Advanced cirrhosis (ascites)
         (d) Severe coagulopathy
         (e) Gravid uterus
   d. The major disadvantages of diagnostic peritoneal lavage are that it can miss significant retroperitoneal bleeding and isolated hollow viscus perforation.
   e. Procedure
      (1) Diagnostic peritoneal lavage can be performed using either a closed, semi-open, or open technique; if performed during pregnancy, the open technique (or mini-lap) should be done using a supraumbilical approach when the gravid uterus is palpable above the umbilicus.
      (2) Before lavage, the stomach and bladder are decompressed (nasogastric tube and Foley catheter).
      (3) A peritoneal lavage catheter is then placed, and aspiration of free intraperitoneal blood attempted. If blood is aspirated, diagnostic peritoneal lavage is considered positive and is terminated.

(4) If no blood is obtained, 1 L (or 10 mL/kg in children) warmed normal saline or lactated Ringer's is instilled into the peritoneal cavity and allowed to drain back out. This effluent is then sent to the laboratory for analysis.

f. RBC criteria for a positive lavage

(1) ≥100,000 RBCs/mm^3 for blunt trauma and anterior abdominal stab wounds

(2) >5,000 RBCs/mm^3 for stab wounds of the lower chest and abdominal gunshot wounds

(3) The RBC count is the most accurate parameter for evaluating lavage fluid. Other criteria (WBC count, amylase, bile) are less reliable and increase only after a delay of several hours. Presence of food particulate is specific for hollow viscus perforation.

G. Pelvic and extremity injuries

1. Because pelvic injuries are frequently encountered in major blunt trauma cases, an AP pelvic radiograph is part of the standard trauma evaluation.
2. During primary survey, extremity evaluation is limited to identifying (and treating) exsanguinating hemorrhage as well as assessing perfusion. It is only during the secondary survey that a more detailed evaluation of the extremities is performed.
3. Once all life-threatening problems have been identified and treated, known and suspected fractures should be immobilized with splints and/or traction, as appropriate, to control pain and bleeding, as well as prevent further injury.
4. After appropriate radiographic evaluation, dislocations should be rapidly reduced.
5. Distal neurovascular function should be assessed promptly and reassessed before and after all extremity manipulations (eg, joint reduction, application of splints, and/or traction).
6. If open wounds/fractures are identified, the patient's tetanus immune status must be assessed and updated as appropriate.
7. Systemic antibiotics should be administered to patients with open fractures and contaminated wounds.
8. Fractured bones bleed extensively. Be prepared to transfuse packed RBCs.
9. **Compartment syndromes can occur later, especially in the lower leg and forearm.**

a. Classic signs of a compartment syndrome (the "6 P's")

(1) Pain out of proportion to the injury and increased with passive stretching and active contraction of the involved muscles

(2) Paresthesias

(3) Paralysis

(4) Pallor

(5) Palpable tenseness and tenderness of the compartment

(6) Pulselessness (a very late finding)

b. Pain is the earliest finding and is followed by the development of paresthesias; all the others are late findings. (For specific bony injuries and further discussion of compartment syndrome, see Orthopedic Emergencies, pages 440–491.)

V. UNIQUE SITUATIONS

A. Electrical injury

1. **Three major insults result from electrical injury.**
   a. **Conduction system changes secondary to damage of the body's electrical system**
      (1) **Cardiac conduction (asystole, ventricular fibrillation, or other cardiac dysrhythmias)**
         (a) **Ventricular fibrillation is the most common cause of death in the acute phase of electrical injuries.**
         (b) **Asystole is the most common arrhythmia in lightning strikes.**
      (2) **CNS conduction (respiratory problems, apnea, seizures, etc) occurs because the body's electrical system is the preferential path for current conduction (it has the least resistance).**
   b. **Thermal tissue damage**
      (1) **The passage of current generates heat → cutaneous burns and muscle injury (conversion of electrical energy to thermal energy is proportional to the resistance of the tissue, ie, higher resistance results in more heat); tissue resistance is greatest in bone, followed in order by skin, muscle, and nerves.**
      (2) **Thermal burns are proportional to the time of exposure (lightning strikes deliver an incredible amount of energy for a very short time period), whereas AC electrical injuries deliver much less energy for a longer period of time.**
      (3) **Muscle injury → rhabdomyolysis → acute myoglobinuric renal failure (if untreated)**
   c. **Blunt trauma**
      (1) **An electric shock can throw the victim down or into the air → fractures (particularly long-bone fractures and spinal compression fractures) and dislocations**
      (2) **An electric shock delivered at a frequency of 40–110 Hz → tetanic contractions → scapular fractures and shoulder dislocations**
2. Classification
   a. Amount of voltage
      (1) Low voltage: <1,000 volts (most household electric sources are 110 or 220V)
      (2) High voltage: >1,000 volts (high-tension power lines may be 100,000 volts; lightning may be in excess of 10 million volts)
   b. Type of electricity
      (1) AC: may cause repetitive muscle contractions and muscle tetany; may actually cause hand to grasp electrical source and prolong exposure
      (2) DC: generally causes a single muscle spasm that throws victim away from source; results in shorter duration of electrical current but more associated trauma from fall
   c. Type of injury
      (1) Direct injury: electrical current passes through the body, which acts as a circuit; there are usually entrance and exit wounds; the internal injuries are often much more severe than the skin wounds would suggest
      (2) Flash (arc) injury: current strikes the skin but does not enter the body; with flash lightning strikes, the classic finding is Lichtenberg figures (branching, "feathering" skin lesions)

3. Management
   a. CPR should be initiated and continued despite prolonged periods of non-perfusing rhythms (usually ventricular fibrillation or asystole), because return of circulation and good neurologic outcomes are more common in victims of electrical injuries.
   b. Patients may have paralysis of respiratory muscles despite a perfusing rhythm.
   c. Victims of lightning strikes may have dilated, nonreactive pupils; these findings do not necessary reflect neurologic status.
   d. Management focuses on ABCs: IV, oxygen, cardiac monitor, cervical spine precautions (if indicated), management of cardiac arrhythmias according to standard ACLS/ATLS protocols, fluid resuscitation
   e. Begin fluid resuscitation with normal saline or lactated Ringer's.
      (1) If there are signs of shock (hypotension/tachycardia, diaphoresis, altered level of consciousness), a fluid bolus (20 mL/kg) is indicated.
      (2) The rate should then be adjusted as needed to maintain a urine output of at least 0.5–1 mL/kg/hr (placing a Foley catheter is helpful).
      (3) If there is evidence of myoglobinuria, the rate should be increased until urine output is 1.5–2 mL/kg/hr.
   f. Order creatine kinase and urine for myoglobin as well as serum potassium and calcium levels (to exclude hyperkalemia and hypocalcemia).
   g. Obtain an ECG on all patients.
   h. Check carefully for entrance and exit wounds; most common entrance sites are the hand and skull; most common exit site is the heel.
   i. Evaluate patients for evidence of blunt trauma.
   j. Cleanse cutaneous burns, and apply a topical antibiotic dressing (eg, silver sulfadiazine, mafenide acetate).
   k. Administer tetanus prophylaxis as needed.
   l. Check for compartment syndromes.
   m. Never debride soft-tissue injuries involving the hands, digits, or face in the emergency department.
   n. Evaluate patients who sustain electrical injuries of the head and neck for traumatic cataracts, and refer them to an ophthalmologist for follow-up; cataracts may develop shortly after injury, or they may be delayed months to years.
   o. Lip burns require close observation and referral to a plastic or oral surgeon; delayed hemorrhage (3–14 days after injury) occurs in 10%–15% of these patients.
4. Disposition
   a. Hospitalization is indicated for all patients with high-voltage burns (>1,000 volts) and for those patients with low-voltage burns (<1,000 volts) who are symptomatic (eg, dysrhythmias, chest pain, cutaneous findings, abnormal urine).
   b. Asymptomatic patients with low-voltage injuries may be discharged to home after a period of observation and cardiac monitoring in the emergency department if their ECG and physical examination are normal and if they have no evidence of cutaneous involvement or urinary heme pigment.
5. Important differences between electrical injuries and thermal burns
   a. Electrical injuries cause extensive muscle damage → myoglobin release (they are more like a crush injury than a thermal burn)

b. In electrical injuries, the extent of cutaneous injury in no way correlates with the amount of underlying tissue damage (neurovascular and musculoskeletal); it is often just the tip of the iceberg. (For further discussion of electrical injuries, see the chapter on Environmental Disorders, page 818.)

6. Lightning injuries: a small but important subset of electrical injuries; see the chapter on Environmental Disorders, page 821.

B. Rhabdomyolysis

1. Pathophysiology: injury to muscle → release of myoglobin → damage to kidneys → acute renal failure (90% of patients recover)
2. Commonly associated electrolyte abnormalities are hypocalcemia (63%) and hyperkalemia (40%).
3. Myoglobin is difficult to assay in serum and is only transiently found in urine, so serial measurements of the creatine kinase level are used to monitor progress.
4. Management
   a. Most important aspect: aggressive IV fluid (normal saline or lactated Ringer's) hydration; administer fluids at a rate sufficient to maintain urine output at 1.5–2 mL/kg/hr.
   b. If this does not clear the pigment, administer mannitol at 25 g IV initially and then add 12.5 g to each subsequent liter of fluid and titrate to maintain urine output >50 mL/hr until the myoglobin is cleared. Furosemide (2 mg/kg IV) may also be administered to promote diuresis.
   c. Alkalinization of the urine with sodium bicarbonate is also helpful. It increases the solubility of myoglobin and facilitates its excretion; 44 mEq of $NaHCO_3$ may be added to each liter of IV fluid.

C. Thermal burns

1. As with all traumatized patients, start with the ABCs. Airway patency and fluid resuscitation are of prime importance.
2. Inhalation injury should be considered in patients with facial burns, singed facial and nasal hair, evidence of oropharyngeal inflammation, carbon deposits in the oropharynx, carbonaceous sputum, and/or a history of fire exposure in a confined space; treat with humidified oxygen and early intubation.
3. Early intubation is indicated in patients with:
   a. Circumferential burns of the neck
   b. Suspected airway injury
4. Early burn resuscitation requires fluid replacement based on the percent of body burned.
   a. Estimate the percent of body surface area (BSA) burned (second- and third-degree burns only) using one of the following:
      (1) "Rule of Nines": head 9%, each arm 9%, chest, back, and each leg 18%, perineum 1%
      (2) "Rule of Palms": surface area of victim's palm is ~1% of his or her BSA
      (3) Lund and Browder charts: more precise than the other two methods, particularly in children
   b. There are many fluid replacement formulas; a general rule is to administer a crystalloid solution (eg, lactated Ringer's) in an amount equal to 2–4 mL/kg/% BSA burned/24 hours. In infants and small children, daily maintenance fluids should also be administered.

(1) Parkland formula: % total BSA × weight (in kg) × 4

(a) Give the first half of these fluids (plus maintenance fluid rate) over the initial 8 hours (from time of burn).

(b) Give the second half of these fluids (plus maintenance fluid rate) over the next 16 hours.

(2) Adjust fluid administration as needed based on urine output (goal 0.5–1 mL/kg/hr), heart rate, and mentation.

For a more detailed discussion of burns and their management, see the chapter on Environmental Disorders, pages 813–823.)

5. Indications for transfer to burn center (American Burn Association Criteria)
   a. ≥25% total BSA partial-thickness burns (≥20% in children <10 years old and adults >40 years old)
   b. ≥10% total BSA full-thickness burns
   c. Burns involving eyes, ears, face, hands, feet, perineum, and any others that may result in significant cosmetic or functional disability
   d. Presence of associated major trauma or inhalation injuries
   e. High-voltage electrical burns

D. Trauma in pregnancy

1. Introduction
   a. Pregnant patients are fundamentally two patients.
   b. Trauma is the most common cause of nonobstetric maternal death during pregnancy.
   c. Because fetal survival depends entirely on maternal integrity, initial management should be directed at resuscitation and stabilization of the mother; prevention of maternal hypoxia and hypotension ensures the best outcome for both mother and fetus.
   d. Early obstetrical, pediatric, and trauma service consults should be obtained.
2. Airway and breathing
   a. Use continuous pulse oximetry.
   b. Administer supplemental 100% oxygen by mask; because fetal blood functions in a lower portion of a left-shifted oxygen–hemoglobin dissociation curve, an increase in oxygen tension produces a significant increase in fetal blood saturation.
   c. If intubation is required in the breathing patient, rapid-sequence intubation (with thiopental, ketamine, or etomidate) using the Sellick maneuver and neuromuscular blockage (with succinylcholine [consider a low-end dose because pseudocholinesterase levels fall in pregnancy] or rocuronium) is recommended.
   d. Thoracostomy tube placement (when indicated) should be at the third or fourth intercostal space to avoid diaphragmatic injury.
3. Circulatory status: hemodynamic parameters are misleading.
   a. An increased heart rate and decreased blood pressure (classic signs of shock) may reflect the normal physiologic changes that occur in association with pregnancy or supine positioning.
   b. Total blood volume increases by 30%–50% during normal pregnancy.
      (1) As a result of this, a pregnant woman may lose 30%–35% of her blood volume (up to 1.5 L) before displaying clinical signs of hypovolemia.
      (2) Maternal blood loss of this volume results in decreased uterine blood flow and fetal hypoxia. Thus, the fetus may be in shock despite the presence of normal maternal vital signs.

c. **Positioning of the pregnant patient on her back → the supine hypotension syndrome, wherein the uterus compresses the inferior vena cava → decreased preload and cardiac output. To prevent this, patients >20 weeks gestation should *be positioned on the left side*. If a spinal injury is suspected, the backboard should be tilted 15° to the left.**

d. Lactated Ringer's solution should be used for initial resuscitation; normal saline may cause hyperchloremic acidosis.

4. Obstetric evaluation is conducted during the secondary survey and it should include:
   a. Fundal height and tenderness
   b. Uterine contractions
   c. Fetal movement
   d. Fetal heart rate (<120 or >160 beats per minute indicates fetal distress, which may be the first sign of maternal hemodynamic compromise)
   e. Inspection of the vulva and outer vaginal vault for blood and secretions (ferning and blue discoloration of nitrazine paper may aid in distinguishing alkaline amniotic fluid from urine)
   f. Pelvic (and rectal) examinations should be performed.
      (1) Perform a careful sterile speculum examination: inspect the cervix for dilation, effacement, and cloudy white or green fluid coming from the os (suggests prolapse of the umbilical cord, which is an obstetric emergency requiring immediate cesarean section).
      (2) Perform a bimanual examination (unless premature rupture of membranes is suspected) to determine fetal station, presence of contractions, or evidence of injury.
5. Patients with gestations >20 weeks should undergo continuous cardiotocographic monitoring. The fetus may be in jeopardy, even with minor maternal injury. Uterine irritability, ruptured membranes, vaginal bleeding, abnormal fetal heart tones/rate (repetitive decelerations, absence of accelerations, beat-to-beat variability) or significant maternal injuries require immediate obstetric consult and admission to the hospital. Usually 4–6 hours of monitoring is recommended.
6. A gestational age >24 weeks is considered to be viable (uterine fundus >3–4 cm/2–3 fingerbreadths above the umbilicus). Estimate gestational age by fundal height or by ultrasonography (femur length or biparietal diameter).
7. **Traumatic uterine rupture may present as maternal shock, or there may be only minimal signs and symptoms. Ultrasonography is very sensitive in detecting uterine rupture and is the diagnostic modality of choice if the equipment and expertise are immediately available. Abdominal radiographic findings include:**
   a. Free intraperitoneal air
   b. Extended fetal extremities
   c. Abnormal fetal position
8. **Placental abruption (abruptio placenta) is the most common cause of fetal death after blunt trauma. Late in pregnancy, abruption can occur after relatively minor injuries.**
   a. Clinical presentation
      (1) External vaginal bleeding (absent in up to 30% of cases)
      (2) Abdominal cramps/pain
      (3) Uterine tenderness/rigidity
      (4) Expanding fundal height

(5) **Maternal shock**

(6) **Fetal distress**

b. **Diagnostic evaluation**

(1) **Cardiotocographic monitoring (most sensitive modality)**

(2) **Uterine ultrasonography**

c. Because abruption may cause disseminated intravascular coagulation, clotting studies should be performed.

9. **Abdominal trauma during pregnancy can result in fetomaternal hemorrage. In an Rh-negative woman carrying an Rh-positive fetus, this can result in isoimmunization if not detected and treated. Thus, all Rh-negative women who sustain abdominal trauma during pregnancy should be given Rh immune globulin.** In women who are <12 weeks pregnant a "mini-dose" (50 mcg) is appropriate; for women >12 weeks pregnant, the standard dose (300 mcg) should be administered. Because Rh immune globulin can effectively prevent Rh isoimmunization when administered within 72 hours of antigenic exposure, the result of the Kleihauer-Betke test are not immediately needed in the emergency department.

10. Perimortem cesarean section

a. Restoration of maternal circulation and thus fetal circulation is the optimal management goal. However, prolonged maternal resuscitation without improvement in maternal circulation may jeopardize the life of a potentially viable fetus. In this situation, decision to perform a perimortem cesarean section must be considered

b. Goal is to deliver fetus within 5 minutes of loss of maternal vital signs. Beyond 20 minutes, there is virtually no chance of survival or a favorable neurologic outcome for the fetus.

11. Prevention: use of seat belts significantly decreases the risk of serious and fatal maternal injuries; properly worn, three-point restraints (lap and shoulder) are the safest.

E. Penetrating trauma

1. Most penetrating injuries result from gunshot and stab wounds (rather than impalements); all penetrating wounds require surgical evaluation.

2. Gunshot wounds

a. Bullets and other missiles (shot) can ricochet and cause much more extensive damage than is visible externally.

b. Gunshot wounds to the lower thorax may involve the abdomen as well. The intraperitoneal cavity extends from the fourth intercostal space anteriorly to the sixth seventh intercostal space posteriorly and laterally.

c. **Gunshot wounds of the abdomen are associated with a high incidence of peritoneal cavity penetration and intraperitoneal injuries; most require immediate laparotomy.**

3. Stab wounds

a. Injuries produced by stab wounds are confined to the path of the instrument.

b. **Stab wounds of the abdomen have a relatively low incidence of intraperitoneal injuries (when compared with gunshot wounds) and hence can be managed selectively; in a patient with stable vital signs, local wound exploration is followed by diagnostic laparoscopy.**

c. Stab wounds to the neck that penetrate the platysma muscle should be extensively evaluated, either surgically in the operating room or with further diagnostic evaluation (chest radiograph, CT, bronchoscopy, esophagogastroduodenoscopy or barium swallow, CT angiography or color Doppler ultrasound).

(1) The best appropriate approach is not known, and most protocols are institutional-dependent; admission for observation is also indicated.

(2) Surgical zones of the neck

(a) Zone I: clavicle to cricoid

(b) Zone II: cricoid to angle of mandible

(c) Zone III: angle of mandible to base of skull

F. Blast injuries

1. Types and mechanisms of injury

a. Primary blast injuries

(1) The result of a pressure wave (caused by an explosion) as it passes through the body. Distance from the blast source determines magnitude of exposure.

(2) The ears, lungs, and GI tract are the most susceptible to injury (tissues with air-fluid interfaces). Commonly associated clinical findings include:

(a) Tympanic membrane rupture (most sensitive indicator of a primary blast effect)

(b) Air emboli on funduscopic exam

(c) Petechial hemorrhages in the nasopharynx

(3) Damage mechanisms

(a) Spallation: occurs when a pressure wave passes from a dense to a less dense medium; results in particles of fluid to be thrown from a dense medium and fragmented into a less dense medium as the pressure wave (eg, explosion in water causes water on surface to splash into air); this affects organs containing both liquid and gas, eg, lungs.

(b) Implosion: compression of gas pockets are produced by the pressure wave (such as occurs in the ear causing tympanic rupture, hearing loss, etc).

(c) Pressure differentials: refer to the difference in pressure that develops between the skin and internal organs at the moment of impact of the shock wave; in the lung, this pressure differential can contribute to pulmonary hemorrhage.

b. Secondary blast injuries are the result of rapid acceleration of small fragments (flying debris, glass, etc) caused by the explosion.

c. Tertiary blast injuries are the result of the victim's collision with a hard surface (frequently a lethal event).

d. Quaternary blast injuries are all explosion-related injuries not classified above; includes the exacerbation of existing conditions, eg, chest pain in a patient with known coronary artery disease.

2. Specific injuries

a. Head and neck are the areas most vulnerable to injury.

(1) Brain injury (primarily subarachnoid hemorrhage)

(2) Skull fracture

(3) Ocular injury (common)

(4) Auditory injury

(a) Hearing impairment

i. May be conductive, sensorineural, or both

ii. Very common in bomb victims (which may lead to an inaccurate assessment, because they are unable to hear verbal commands and respond appropriately)

iii. If associated with brain injury, skull fracture, vertigo, or dizziness, suspect ossicular damage.

(b) Tympanic membrane peforation

i. Mechanisms of injury

- Inertia and pressure differentials → linear or small perforations
- Spalling → large punched-out perforations

ii. Usual site of injury → the pars tensa of the tympanic membrane

(5) Facial injury → spalling in the ethmoid region → anosmia

b. Lungs

(1) Pulmonary edema and hemorrhage (most common in the intercostal spaces) are the predominant findings

(2) Earliest signs: cough, tachypnea, hypoxemia ("blast lung syndrome")

(3) ARDS may occur 24–48 hours after the blast.

(4) Airway management

(a) Unassisted ventilation (with the patient on oxygen) is best.

(b) Assisted ventilation (with the patient intubated) carries a risk of tension pneumothorax; in addition, therapy with positive end-pressure ventilation should be avoided, because there is potential risk of air embolism associated with its use in these patients.

(5) General anesthesia should be delayed 1–2 days (if possible), because it is poorly tolerated in patients with primary blast injury; however, regional or spinal anesthesia may be used.

c. GI tract

(1) Air-containing organs (stomach, duodenum, colon) as well as solid viscera (liver, spleen, diaphragm) may rupture.

(2) Delayed perforations may also occur (especially in the ileocecal region).

(3) Intestinal injuries are common in underwater blasts.

G. Additional pearls

1. The spleen is the most common abdominal organ injured in blunt trauma, followed by the liver.
2. The liver is the most common abdominal organ injured in penetrating trauma, followed by the small bowel.
3. The most common cause of sudden death after a motor vehicle collision or fall from a great height is traumatic aortic rupture.
4. A sternal fracture or chest wall contusion may be associated with a potentially more serious injury, eg, myocardial contusion, traumatic aortic dissection.
5. Pelvic fractures are associated with bladder injury; check for hematuria and do a urethrogram/cystogram if appropriate.
6. Posterior knee dislocations may be associated with injury to the popliteal artery and/or nerve; arteriography may be indicated (after surgical consult) even if pulses are present. If you are skilled in joint reduction, you may attempt one gentle reduction maneuver.
7. Solitary lap belts are associated with small-bowel injuries and mesenteric lacerations.
8. Up to 20% of patients with severe renal trauma do not have hematuria; if there is a high index of suspicion, perform a CT scan.

9. The most common injury likely to present late is a contused bowel (eg, duodenal hematoma); initial CT may be normal.
10. Continuous reevaluation (eg, serial abdominal examinations) of the trauma patient is important.

TRAUMATIC DISORDERS: PRACTICE CLINICAL SCENARI

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A patient has sustained blunt thoracic trauma and presents with tachypnea and retractions.

Physical examination: Pulse oximetry is 90% on 100% oxygen. Primary survey reveals a patent airway but decreased breath sounds on the affected side. The tra is deviated to the contralateral side. The ipsilateral chest is hyperexpanded and hyperresonant on percussion.

What is the diagnosis?

Scenario B

Presentation: A 30-year-old man sustains a closed head injury from a 30-foot fal had a brief period of loss of consciousness immediately after the fall. He is now but deteriorating, and develops confusion, decreased level of consciousness, an coma.

What is the diagnosis?

Scenario C

Presentation: A patient arrives at the emergency department after a high-speed r vehicle collision in which he was unrestrained and possibly ejected from the ve He is tachycardic and hypotensive.

Physical examination: The airway is secured, and no obvious signs of thoracic ir are found. A FAST exam reveals fluid in Morison's pouch.

What is the diagnosis?

Scenario D

Presentation: An elderly gentleman who is on supplemental oxygen at home pr after sustaining a flash burn while trying to light his cigarette. He is awake, alert talking, and has partial-thickness burns to his entire face. His nose hairs are sing and there is soot in his oropharynx. His oxygen saturation is 95% on a nonrebre mask.

What is the diagnosis?

Scenario E

Presentation: A 14-year-old presents with abdominal and back pain after a motor vehicle collision in which he was wearing only a lap belt, and the car rear-ended another vehicle at high speed.

Physical examination: The patient is hemodynamically stable. On examination, there is a linear ecchymotic bruise or abrasion across the mid-abdomen, and the abdomen is tender. There is point thoracolumbar midline tenderness.

What is the diagnosis?

Scenario D

Diagnosis: burn with inhalational injury

Diagnostic evaluation: The key to diagnosis is recognizing that this patient has an inhalation injury and is at very high risk of developing airway compromise and respiratory failure.

Management: This is an airway emergency. This patient should be intubated immediately. A difficult airway should be anticipated, and advanced airway equipment, airway alternatives, and surgical airway equipment readily available. After intubation, partial-thickness burns should be treated by standard practice with fluid resuscitation, debridement of nonviable tissue, and wound cleansing. The patient should be transferred to a burn center.

Scenario E

Diagnosis: lap belt syndrome

Diagnostic evaluation: The key to diagnosis is recognizing that this mechanism of injury combined with the examination findings is very suspicious of "lap belt syndrome" (abdominal wall hematoma/bruise, thoracolumbar Chance fracture, and small-bowel injury). FAST examination will likely be negative, because this is a hollow- rather than solid-organ injury. CT is most sensitive but may still not detect some hollow viscus injuries. Imaging of the thoracolumbar spine is essential.

Management: Intra-abdominal injuries may be missed on initial evaluation, in which case, observation and serial abdominal examinations are essential. They are usually managed by exploratory laparotomy. Thoracolumbar Chance fractures are unstable. Spinal immobilization is essential during emergency department evaluation. These fractures are usually stabilized neurosurgically in the operating room.

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: tension pneumothorax

Diagnostic evaluation: The diagnosis should be made immediately based on clinical presentation. No laboratory or radiologic studies should be performed.

Management: Immediate needle thoracostomy should be performed on the affected side, using a large bore over-the-needle catheter. The needle is inserted over the third rib (second intercostal space) perpendicular to the chest. A gush of air may be heard. After successful decompression, a tube thoracostomy should be performed.

Scenario B

Diagnosis: epidural hematoma

Diagnostic evaluation: The diagnosis should be suspected based on the clinical history. It is confirmed by noncontrast head CT, which shows a classic lens-shaped convex hematoma, usually along the temporal bone.

Management: Airway should be secured before head CT if the patient shows signs of neurologic deterioration. Standard management of increased ICP should be implemented. In the setting of acute herniation, hyperventilation is the most rapid measure to decrease ICP. Ultimately, neurosurgical decompression is definitive care. Consultation should be made early in course.

Scenario C

Diagnosis: intra-abdominal injury resulting in hemorrhagic shock

Diagnostic evaluation: The key to diagnosis is identifying hemorrhagic shock (tachycardia and hypotension) and performing a FAST examination. The unstable patient should not leave the resuscitation bay for a CT.

Management: Management in the emergency department focuses on restoring circulatory volume and oxygen-carrying capacity. Place two large-bore IV lines, and administer crystalloid fluids, 1–2 L over 5 minutes each. If the patient remains in shock, administer packed RBCs. Ultimately the unstable patient with intra-abdominal bleeding needs an exploratory laparotomy in the operating room. Consultation with trauma or general surgery early in the course is essential.

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ORTHOPEDIC EMERGENCIES: SELF-ASSESSMENT QUESTIONS

1. All the following statements regarding anterior shoulder dislocations are true except:
   (a) Subcoracoid dislocations are the most common type.
   (b) Associated neurovascular injury is nonexistent.
   (c) A Hill-Sachs deformity is seen in up to 50% of anterior dislocations.
   (d) Treatment consists of reduction and immobilization in a shoulder immobilizer.

2. Which of the following statements regarding posterior shoulder dislocations is accurate?
   (a) They are best visualized with an axillary or scapular "Y" view.
   (b) They are often associated with damage to the axillary nerve.
   (c) They are the most frequent type of shoulder dislocation.
   (d) They are often associated with fracture of the anterior glenoid rim.

3. Which of the following is not a component of the rotator cuff?
   (a) Subscapularis
   (b) Supraspinatus
   (c) Infraspinatus
   (d) Teres major

4. All of the following statements regarding the rotator cuff are true except:
   (a) It permits abduction and controls internal and external rotation of the shoulder.
   (b) The tendinous insertions of the rotator cuff are on the greater and lesser tuberosities of the humerus.
   (c) It may be torn in association with anterior shoulder dislocations.
   (d) All tears require surgical correction.

5. All of the following statements regarding humeral fractures are accurate except:
   (a) Humeral shaft fractures are frequently associated with radial nerve injuries.
   (b) Proximal humeral fractures are classified according to the Neer classification system.
   (c) Fracture through the surgical neck of the humerus is associated with avascular necrosis of the humeral head.
   (d) Management of proximal humeral fractures is determined primarily by the amount of displacement present.

6. All of the following statements regarding supracondylar extension fractures are true except:
   (a) They generally result from a fall on the outstretched arm.
   (b) They are more common in adults than in children.
   (c) They are associated with the development of Volkmann ischemic contracture.
   (d) Associated median nerve injury may occur.

7. A distal radial shaft fracture associated with a distal radioulnar dislocation is referred to as a _____.

   (a) Galeazzi fracture
   (b) Monteggia fracture
   (c) Nightstick fracture
   (d) Maisonneuve fracture

8. All the following statements regarding the ulnar nerve are accurate except:

   (a) It passes through Guyon canal.
   (b) Loss of function results in a "claw hand" deformity.
   (c) It innervates the interosseous muscles.
   (d) It is frequently injured in association with anterior elbow dislocations.

9. The carpal bone most frequently injured in a fall on the outstretched hand is the _____.

   (a) Lunate
   (b) Trapezoid
   (c) Scaphoid
   (d) Triquetrum

10. The incidence of avascular necrosis in association with scaphoid fractures is highest in fractures of the _____.

    (a) Tubercle of the scaphoid
    (b) Distal scaphoid
    (c) Waist (middle third) of the scaphoid
    (d) Proximal scaphoid

11. Which of the following injuries is most frequently associated with the development of acute carpal tunnel syndrome?

    (a) Perilunate dislocation
    (b) Lunate dislocation
    (c) Scapholunate dislocation
    (d) None of the above

12. All but one of the injuries listed below may be sustained in a fall on the outstretched arm. Which of them occurs via a different mechanism?

    (a) Rotator cuff tear
    (b) Proximal humeral fracture
    (c) Radial head subluxation
    (d) Supracondylar fracture

13. The primary cause of death in patients with pelvic fractures is _____.

(a) Blood loss
(b) Associated injuries
(c) Ruptured diaphragm
(d) Sepsis

14. All of the following statements regarding posterior hip dislocations are true except:

(a) They represent 80%–90% of all hip dislocations.
(b) Avascular necrosis is a late complication of this injury.
(c) The limb appears abducted, externally rotated, and flexed.
(d) The mechanism of injury is a direct force applied to the flexed knee.

15. All of the following are signs of pelvic fracture except:

(a) Destot sign
(b) Earle sign
(c) McMurray sign
(d) Roux sign

16. Which of the following are stabilizers of the lateral aspect of the knee?

(a) Vastus lateralis and rectus femoris muscles
(b) Semimembranosus muscle and medial collateral ligament
(c) Iliotibial band and biceps femoris muscle
(d) Cruciate ligaments

17. All of the following statements regarding knee dislocations are accurate except:

(a) The incidence of associated popliteal artery injury is 30%–40%.
(b) Findings of a warm foot and palpable pulses exclude the presence of injury to the popliteal artery.
(c) Findings of paresthesia along the dorsal aspect of the foot and foot drop signal the presence of associated peroneal nerve injury.
(d) Vascular assessment is indicated for all patients with this injury.

18. Which of the following is not an early or reliable sign of anterior compartment syndrome?

(a) Pain with active dorsiflexion of the foot
(b) Sensory loss in the first web space of the foot
(c) Absence of peripheral pulses of the foot
(d) Pain with passive plantar flexion of the foot

19. Which of the following findings is consistent with a third-degree sprain of the ankle?

    (a) Moderate swelling
    (b) Ability to bear weight
    (c) Positive stress test
    (d) Moderate functional loss

20. All of the following statements regarding calcaneal fractures are true except:

    (a) They have a 10% incidence of associated lumbar fractures.
    (b) They are generally the result of a compression injury (fall).
    (c) They are the second most frequently fractured tarsal bone.
    (d) A Bohler angle <20° is consistent with a depressed fracture of the body of the calcaneus.

21. The joint separating the hindfoot from the midfoot is referred to as:

    (a) Charcot joint
    (b) Chopart joint
    (c) Lisfranc joint
    (d) Bohler joint

22. All of the following statements regarding lunate fractures are accurate except:

    (a) Patients present with pain over the mid-dorsum of the wrist that is increased by axial compression of the third metacarpal.
    (b) Plain radiographs are usually diagnostic.
    (c) They result from a fall on the outstretched hand.
    (d) Avascular necrosis of the proximal segment is a serious complication.

23. A 28-year-old athlete presents with knee pain. He states that he was playing football about 2 hours ago and received a direct blow to the lateral aspect of his knee. Radiograph of the knee is negative for fracture; examination reveals a hemarthrosis. Ligamentous evaluation reveals instability (joint opening of 5 mm) with valgus stress in 30° of flexion. Other tests of stability, including valgus stress in extension, are negative. This athlete most likely has:

    (a) Rupture of the anterior cruciate ligament
    (b) Rupture of the medial collateral ligament
    (c) Rupture of the medial collateral ligament as well as potential injury to the anterior cruciate ligament and posterior capsule
    (d) Rupture of the lateral collateral ligament

24. A 60-year-old woman presents with the complaint of knee pain for one day. There is no history of trauma or similar pain in the past. Examination reveals a tender, warm, erythematous knee. Joint aspiration demonstrates cloudy fluid with 10,000 WBCs (>75% PMNs), normal glucose, and rhomboidshaped crystals that are positively birefringent under polarized light. The most likely diagnosis is:

    (a) Osteoarthritis
    (b) Rheumatoid arthritis
    (c) Gout
    (d) Pseudogout

25. A 40-year-old patient presents with low back pain plus bowel and bladder incontinence. Examination reveals loss of sensation in the "saddle" distribution and loss of anal tone. The most appropriate management of this case is:

    (a) Bed rest for 2 days with appropriate analgesia and muscle relaxants plus orthopedic follow-up in 2 days.
    (b) Bed rest for 2 weeks with appropriate analgesia and muscle relaxants plus orthopedic follow-up in 2 weeks.
    (c) Immediate neurosurgical consult for acute disc decompression.
    (d) Bed rest for 2 days with appropriate analgesia and muscle relaxants plus neurosurgical follow-up in 2 days.

26. A 62-year-old woman presents with severe shoulder and proximal arm pain after a fall. Radiograph reveals a proximal humeral fracture; the lesser tuberosity is displaced from the remainder of the humerus by >l cm. This type of proximal humeral fracture would most accurately be classified as a Neer _____.

    (a) One-part fracture
    (b) Two-part fracture
    (c) Three-part fracture
    (d) The Neer classification system does not pertain to this type of fracture.

27. Which of the injuries listed below commonly occurs in association with the particular type of humeral fracture described in the question above (#26)?

    (a) Clavicular fracture
    (b) Posterior shoulder dislocation
    (c) Anterior and posterior shoulder dislocations
    (d) Anterior shoulder dislocation

28. All the following statements regarding compartment syndrome are accurate except:

    (a) It can be caused by crush injuries, fractures, or constrictive dressings or casts.
    (b) The most commonly affected compartments are the anterior compartment of the lower leg and the volar compartment of the forearm.
    (c) Normal compartment pressure is 10–20 mmHg.
    (d) Initial management consists of removal of constricting dressings or casts (if present).

29. Which of the following is an absolute contraindication to reimplantation of an amputated digit?

    (a) Single-digit amputation other than the thumb
    (b) Serious underlying systemic illness
    (c) Severely damaged or contaminated part
    (d) Unstable patient with other life-threatening injuries

30. While awaiting a decision from the vascular surgeon regarding reimplantation, what is the best method of preserving an amputated part?

    (a) Place it in a container of 10% povidone-iodine solution, and store this container in ice water.
    (b) Irrigate it with normal saline or lactated Ringer's to remove gross contamination, wrap it in sterile gauze moistened with normal saline, place it in a sterile, water-tight container, and store this container in ice water.
    (c) Irrigate it with normal saline or lactated Ringer's to remove gross contamination, wrap it in sterile gauze moistened with normal saline, and then place it on ice.
    (d) Irrigate it with 10% povidone-iodine solution to remove gross contamination, wrap it in sterile gauze moistened with normal saline, and place it between two ice packs.

31. All of the following statements regarding scapular fractures are accurate except:

    (a) They typically occur in association with high-energy trauma.
    (b) Associated injuries, some of which may be life- or limb-threatening, occur in up to 98% of these patients.
    (c) The most common associated injuries are ipsilateral lung injuries, rib fractures, and clavicle fractures.
    (d) Most scapular fractures require orthopedic referral for open reduction and internal fixation.

32. The most commonly injured major ligament of the knee is the _____.

    (a) Medial collateral ligament
    (b) Lateral collateral ligament
    (c) Anterior cruciate ligament
    (d) Posterior cruciate

33. A 48-year-old man presents with knee pain after a fall. Examination reveals a significant hemarthrosis, which you aspirate to make him more comfortable. The finding of fat globules in the aspirate is very suggestive of:

    (a) A tear in the anterior cruciate ligament
    (b) A peripheral meniscus tear
    (c) A fracture
    (d) None of the above

34. Innervation of the intrinsic muscles of the hand is provided by:

(a) The median and ulnar nerves
(b) The ulnar and radial nerves
(c) The median and radial nerves
(d) The median, ulnar, and radial nerves

35. The Apley and McMurray tests are helpful in making the diagnosis of:

(a) Chondromalacia patellae
(b) Meniscal tears
(c) A torn anterior cruciate ligament
(d) A torn posterior cruciate ligament

ANSWERS

1. b	7. a	13. a	19. c	25. c	31. d
2. a	8. d	14. c	20. c	26. b	32. c
3. d	9. c	15. c	21. b	27. b	33. c
4. d	10. d	16. c	22. b	28. c	34. a
5. c	11. b	17. b	23. b	29. d	35. b
6. b	12. c	18. c	24. d	30. b	

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. GENERAL PRINCIPLES OF FRACTURE AND DISLOCATION MANAGEMENT

A. Classification and definitions

1. Direct trauma (direct force over the fracture site)
   a. "Tapping" (nightstick) fracture
      (1) Linear fracture with two fragments
      (2) Little or no soft-tissue injury
   b. Crush fracture
      (1) Comminuted or transverse fracture
      (2) Extensive soft-tissue injury
   c. Penetrating fracture (often seen with missile wounds)
      (1) High-velocity injuries: fragmentation of bone → bone fragments act as secondary missiles → cavitation and extensive soft-tissue injury
      (2) Low-velocity injuries: mild fragmentation of bone
2. Indirect trauma (forces acting at a distance from the fracture site)
   a. Traction (tension) fracture: bone is pulled apart → transverse fracture
   b. Angulation fracture: bending along the long axis of the bone → transverse fracture with concave surface often splintered
   c. Compression fracture: compression on the long axis of the bone → axial loading → "T" or "Y" fractures
   d. Spiral fracture: results from rotational stress (torque)
      (1) A pure rotational injury is rare.
      (2) When associated with an axial load → oblique fracture
3. Incomplete fractures (torus and greenstick): only one cortex is interrupted; these fractures occur almost exclusively in children (because of their greater bone elasticity).
   a. Torus (buckle) fracture: characterized by a bulging (buckling) of one cortex; results from compressive forces and usually involves the metaphyseal region
   b. Greenstick fracture: characterized by a break in one cortex (convex side) and a bending (bowing) of the other cortex (concave side); results from an angular force applied to a long bone
4. Complete fracture: involves both cortices
5. Closed (simple) fracture: no communication with the external environment; overlying skin and soft tissue are intact.
6. Open (compound) fracture: communication with the external environment through a break in the overlying skin and soft tissue; high risk of infection (osteomyelitis); considered an orthopedic emergency. Management includes:
   a. Early emergency department administration of IV antibiotics (a first-generation cephalosporin with or without an aminoglycoside)
   b. Tetanus prophylaxis
   c. Wound irrigation and debridement (usually accomplished in the operating room)
7. Pathologic fracture: results from bone weakness secondary to an underlying disease process (cysts, tumors, osteogenesis imperfecta, scurvy, rickets, Paget disease, etc); should be suspected when trivial trauma results in a fracture

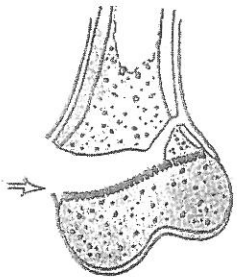
8. Stress fracture ("march or fatigue fracture"): results from bone fatigue secondary to repeated or cyclical stress; often occurs in association with a sudden increase in level of training; usually located in the lower extremities (metatarsals, navicular, distal tibia/fibula, femoral neck); plain radiographs may be negative for ≥2 weeks.
9. Dislocation: displacement of a bone from its normal position so that there is complete disruption of the articular surface
10. Subluxation: a partial dislocation when there is incomplete disruption of the articular surface

B. Salter-Harris classification of epiphyseal fractures (aids in determining prognosis: the higher the number, the poorer the prognosis)

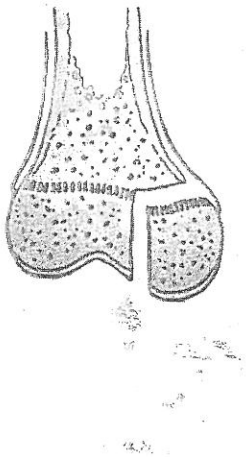
1. **Type I: fracture through the epiphyseal plate (physis), resulting in separation of the epiphysis; good prognosis.**



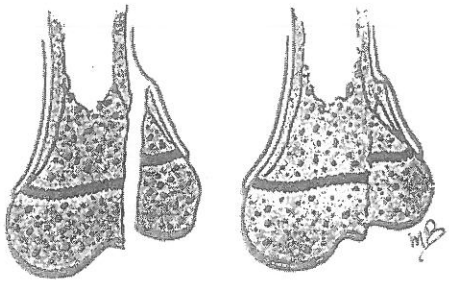
2. **Type II: fracture of the metaphysis with extension through the epiphyseal plate (most common type); most common in children >10 years old; a metaphyseal fragment called a "Thurston Holland sign"' is present; good prognosis.**



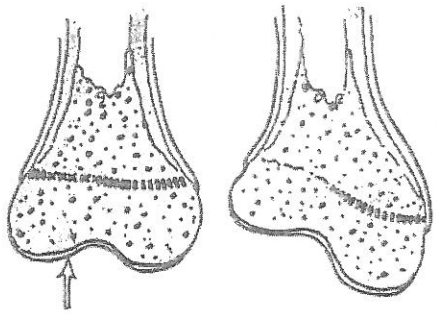
3. **Type III: fracture of the epiphysis with extension into the epiphyseal plate (a totally intra-articular fracture); open reduction is often necessary.**



4. IV: fracture through the metaphysis, epiphysis, and epiphyseal plate (also a comple intra-articular fracture); open reduction is usually necessary, and perfect reduction essential; growth disturbance is common.



5. Type V: crush fracture of the epiphyseal plate (opposite of Type 1): most commonly seen in the knee and ankle; radiographs may look normal; poor prognosis, because the blood supply to the epiphyseal plate is interrupted.



C. Complications of fractures

1. Immediate complications
   a. Hemorrhage (can be extensive with pelvic fractures)
   b. Vascular injuries
      (1) Anterior shoulder dislocation → exclude axillary artery injury
      (2) Extension supracondylar fracture → exclude brachial artery injury
      (3) Posterior elbow dislocation → exclude brachial artery injury
      (4) Knee dislocation → exclude popliteal artery injury
   c. Nerve injuries
      (1) Anterior shoulder dislocation → exclude axillary and musculocutaneous nerve inju
      (2) Humeral shaft injury → exclude radial nerve injury
      (3) Extension supracondylar fracture → exclude median, radial, and ulnar nerve injury
      (4) Medial epicondylar fracture → exclude ulnar nerve injury
      (5) Posterior elbow dislocation → exclude ulnar and median nerve injury
      (6) Olecranon fracture → exclude ulnar nerve injury
      (7) Acetabular fracture → exclude sciatic nerve injury
      (8) Posterior hip dislocation → exclude sciatic nerve injury
      (9) Anterior hip dislocation → exclude femoral nerve injury
      (10) Knee dislocation → exclude peroneal and tibial nerve injury
      (11) Lateral tibial plateau fracture → exclude peroneal nerve injury
   d. Associated soft-tissue and visceral injuries

2. Intermediate complications
   a. Compartment syndrome
   b. Fat embolism (usually originates from a long bone)
3. Long-term complications
   a. Reflex sympathetic dystrophy
   b. Volkmann ischemic contracture
   c. Nonunion
   d. Avascular necrosis (femoral head, proximal scaphoid, capitate, and talus fractures are particularly predisposed to this complication)
   e. Angulation deformities, overgrowth or shortening
   f. Infection
   g. Joint stiffness
   h. Posttraumatic ossification or arthritis

II. UPPER EXTREMITY TRAUMA

A. Shoulder dislocations and ligamentous tears

1. Sternoclavicular joint injuries
   a. Classification
      (1) First degree (sprain) → partial tear of the sternoclavicular and costoclavicular ligaments without associated clavicular subluxation
      (2) Second degree (subluxation) → complete tear of the sternoclavicular ligament plus partial tear of the costoclavicular ligament with subluxation of the clavicle from the manubrium
      (3) Third degree (dislocation) → complete tear of both the sternoclavicular and costoclavicular ligaments with dislocation of the clavicle from the manubrium; clavicle can dislocate either anteriorly (most common) or posteriorly (uncommon, but a true orthopedic emergency).
   b. Mechanism of injury
      (1) Direct force over sternoclavicular joint → posterior dislocation
      (2) Fall onto the shoulder → anterior or posterior dislocation (large amount of force involved, often necessitating trauma evaluation and consideration of possible intrathoracic injuries)
   c. Clinical presentation
      (1) Tenderness and swelling over the sternoclavicular joint
      (2) Pain with movement of the ipsilateral extremity and with lateral compression of the shoulders
      (3) With third-degree injuries, the medial clavicle is displaced (anteriorly or posteriorly) relative to the manubrium.
      (4) Shortness of breath, dysphagia, or choking (in patients with posterior dislocations associated with compression of mediastinal structures)
   d. Diagnostic evaluation: radiographs; dislocations best demonstrated with CT

e. Treatment
   (1) First degree → arm sling for 3–4 days and analgesics
   (2) Second degree → arm sling, analgesics, and orthopedic follow-up
   (3) Third degree → immediate orthopedic consult and rapid reduction
      (a) Establish an IV line and administer analgesia; posterior dislocations often require general anesthesia in the operating room.
      (b) Place the patient in the supine position with a rolled sheet between the shoulders.
      (c) Extend, abduct, and apply traction to the ipsilateral arm while an assistant pushes (anterior dislocation) or pulls with a towel clip (posterior dislocation) the clavicle into its normal position.
      (d) Apply an arm sling, and refer for orthopedic follow-up.
      (e) Life-threatening injuries to adjacent structures (pneumothorax and compression or laceration of the esophagus, trachea, or great vessels) occur in up to 25% of posterior dislocations and must be promptly attended to.

2. Acromioclavicular separation
   a. Classification
      (1) First degree/Type I (sprain) → partial tear of acromioclavicular ligament without subluxation of the clavicle; the coracoclavicular ligament is intact.
      (2) Second degree/Type II (subluxation) → complete tear of acromioclavicular ligament with subluxation of the clavicle; coracoclavicular ligament is stretched or incompletely torn.
      (3) Third degree/Type III (dislocation) → complete tears of both the acromioclavicular and coracoclavicular ligaments with dislocation of the clavicle
      (4) Fourth degree/Types IV–VI (displacement) → significant displacement of the distal clavicle posteriorly (Type IV), superiorly (Type V), or inferiorly (Type VI), as well as associated injury/ interposition of the deltoid or trapezius muscle
   b. Mechanism of injury
      (1) Fall on the shoulder with the arm adducted (most common)
      (2) Fall on the outstretched arm
   c. Clinical presentation
      (1) Tenderness and swelling over the acromioclavicular joint
      (2) Pain with movement of the affected extremity
      (3) With Type III injuries, the distal clavicle is displaced upward relative to the acromion when compared with the opposite shoulder. This is best visualized when the patient is examined in the sitting or standing position with the affected arm hanging at his or her side.
   d. Diagnostic evaluation
      (1) Radiographic evaluation
      (2) Obtain AP views of both clavicles
      (3) Stress views (taken with 10 lbs of weight suspended from each wrist) are no longer routinely recommended because they:
         (a) Are associated with increased cost, radiation exposure, and patient discomfort
         (b) Provide little additional information and can be misleading

(c) Do not usually alter the course of treatment, because most acromioclavicular separations are managed conservatively (nonoperatively), and most third-degree injuries are clinically obvious.

(4) Findings depend on the degree of separation.

(a) First degree → acromioclavicular joint is radiographically normal.

(b) Second degree → due to upward displacement of the clavicle, the distance between the acromion and the inferior aspect of the distal aspect of the clavicle is increased by ≤½ the width of the clavicle (or ≤1 cm) on AP view. The distance seen between the clavicle and the coracoid process is normal.

(c) Third degree → the distance between the acromion and the distal aspect of the clavicle is increased by >½ the width of the clavicle (or >1 cm) on the AP view. The distance between the distal clavicle and the coracoid process is also increased.

e. Treatment

(1) Type I → sling for 1–2 weeks and analgesics, followed by early range of motion exercises

(2) Type II → sling until acute pain has subsided; analgesics and orthopedic referral for further evaluation and rehabilitation

(3) Type III → immobilization versus surgical fixation is controversial; immobilize in a sling, provide analgesics, and arrange prompt orthopedic referral (within 72 hours).

(4) Types IV–VI → will likely require surgical fixation as definitive treatment; acute management is the same as that for Type III.

3. Shoulder dislocation (most common dislocation seen in the emergency department)

a. Diagnostic evaluation: radiographic series

(1) Standard views should always be ordered (AP shoulder and transcapular lateral or "Y" view)

(a) In anterior dislocations, the AP view detects the most important associated fracture—the humeral neck; fracture of the lesser tuberosity suggests a posterior shoulder dislocation.

(b) In posterior dislocations, the "Y" or axillary view is diagnostic.

(2) An axillary lateral view is ideal for differentiating anterior from posterior dislocations; it often reveals an impression fracture of the humeral head, but it may be difficult to obtain.

(3) A modified axillary view (called the West Point view) allows visualization of the anterior glenoid rim; avulsion (Bankart) fractures in this area (that are associated with anterior dislocations) are not infrequent.

b. Anterior (95%–97% of all shoulder dislocations)

(1) Mechanism of injury: abduction, extension, and external rotation

(2) Types (where the humeral head is)

(a) Subcoracoid (most common)

(b) Subglenoid (head of the humerus is anterior and inferior to the glenoid fossa)

(c) Subclavicular (very rare)

(d) Intrathoracic (very rare)

(3) Clinical presentation

(a) Examination of the shoulder reveals prominence of the acromion process and flattening of the normal contour of the shoulder. The affected arm is held in slight abduction and external rotation; the patient is unable to place his or her palm on the uninjured shoulder.

(b) A clinical clue is resistance to internal rotation and adduction.

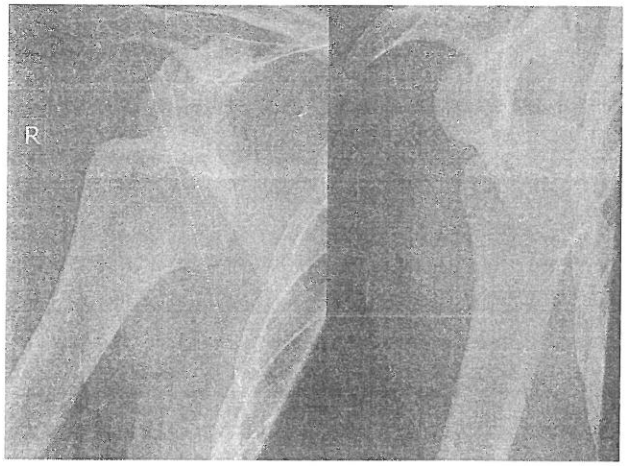
(4) Complications/associated injuries

(a) Recurrence is the most common complication and is age related; the younger the patient, the greater the likelihood of recurrence.

(b) Bony injuries

i. Hill-Sachs deformity

- A compression fracture or "groove" of the posterolateral aspect of the humeral head



Source: Hellerhoff (Wikimedia Commons: http://en.wikipedia.org/wiki/File:Dislocated_shoulder_X-ray_03.png)

- Results from impaction of the humeral head on the anterior glenoid rim as it dislocates (or reduces)
- Occurs in up to 50% of anterior dislocations and is particularly common in patients with recurrent dislocation; the humeral head is damaged by the sharp anterior rim of the glenoid, creating a lesion called the "hatchet sign," which is apparent on the reduction film.

ii. Avulsion of the greater tuberosity (more common in patients >45 years old and in those with subglenoid dislocations)

iii. Fracture of the anterior glenoid lip (Bankart fracture)

(c) Nerve injuries

i. May occur when the shoulder is dislocated or reduced; therefore, it is important to check and document sensation both before and after reduction.

ii. Most injuries are neuropraxias and recover well over time.

iii. Axillary nerve injury (most common): examination reveals sensory loss over the lateral aspect of the shoulder and weakness in shoulder abduction (deltoid muscle).

iv. **Musculocutaneous nerve injury: examination reveals weakness of forearm flexors and supinators, as well as sensory loss along dorsum of forearm.**

v. **Brachial plexus injuries: require atraumatic reduction**

(d) **Rotator cuff tears (higher incidence in patients >40 years old)**

(e) **Axillary artery injury is rare but should be suspected in an elderly patient with a weak/absent radial pulse or an expanding hematoma.**

c. Posterior (2%–4% of all shoulder injuries)

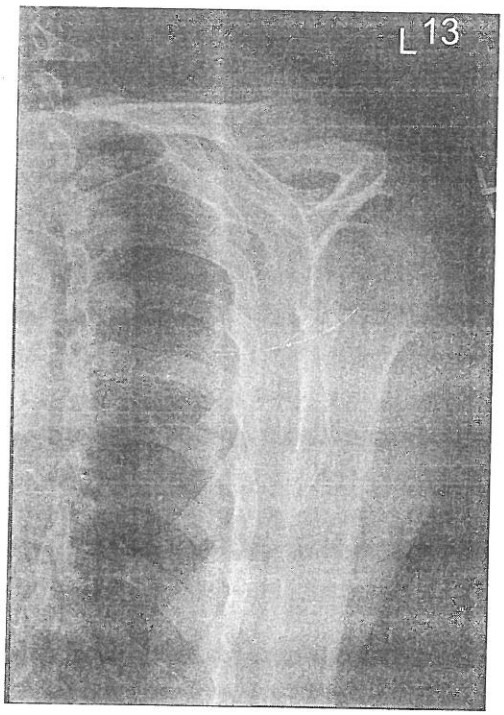
(1) Mechanism of injury

(a) Convulsive seizure or electric shock

(b) Fall on a forward-flexed, adducted, and internally rotated arm

(c) Significant direct blow to the anterior shoulder

(2) The most commonly missed major dislocation of the body, it is often misdiagnosed as "bursitis" or "adhesive capsulitis." Radiographic diagnosis can be made with an axillary lateral or transscapular lateral "Y" view; an axillary lateral view is an excellent technique for visualization of a posterior dislocation, but it is not usually possible to take because the patient cannot abduct his or her shoulder. (Be able to identify.)



Courtesy of Michael C. Bond, MD

(3) Types

(a) Subacromial (98%)

(b) Subglenoid

(c) Subspinous

(4) Clinical presentation

(a) Examination of the shoulder reveals anterior flatness, posterior fullness, and prominence of the coracoid process. The affected arm is internally rotated and is usually held in adduction.

(b) A clinical clue is inability to abduct or externally rotate the arm.

(5) Complications/associated injuries

(a) Associated fractures of the posterior rim of the glenoid fossa, anteromedial aspect of the humeral head (reversed Hill-Sachs deformity), and lesser tuberosity. In fact, in the presence of an isolated lesser tuberosity fracture, a posterior shoulder dislocation should always be excluded.

(b) Recurrence rate is 30%.

(c) Neurovascular complications are uncommon.

d. Treatment of shoulder dislocation is with reduction using traction, scapular manipulation, or leverage and requires procedural sedation. Closed reduction can usually be accomplished in the emergency department.

(1) Reduction techniques for anterior dislocations

(a) Stimson or hanging-weight

(b) Scapular manipulation

(c) Traction-countertraction

(d) External rotation (Hennipen technique)

(e) Milch forward elevation

(f) Spaso maneuver

(g) The Kocher maneuver (a leverage technique) and the Hippocratic technique are associated with many complications and should not be used.

(2) Reduction technique for posterior dislocations

(a) Axial traction is applied in-line with the humerus, and then the humeral head is pushed forward.

(b) If this is unsuccessful, reduction may need to be accomplished under general anesthesia. Orthopedic consult should precede any reduction attempt, because this injury is rare and the rate of complications (fractures) is high.

(3) Shoulder dislocations with an associated fracture should be referred to an orthopedic surgeon for reduction (closed or open).

(4) After reduction

(a) Recheck and document neurovascular examination.

(b) Order radiographs to confirm reduction and exclude any associated injuries; humeral neck fractures are a known complication of anterior shoulder relocation, the result of which is often avascular necrosis of the humeral head.

(c) Place patient in a shoulder immobilizer or Velpeau dressing. The length of immobilization will vary from 1 to 4 weeks, depending on the patient's age; the older the patient, the sooner mobilization should be started to avoid stiffness.

(d) First-time dislocations should be placed in a shoulder immobilizer with the shoulder held at 15° external rotation; this helps to reduce the dislocation from recurring.

(e) Refer for orthopedic follow-up.

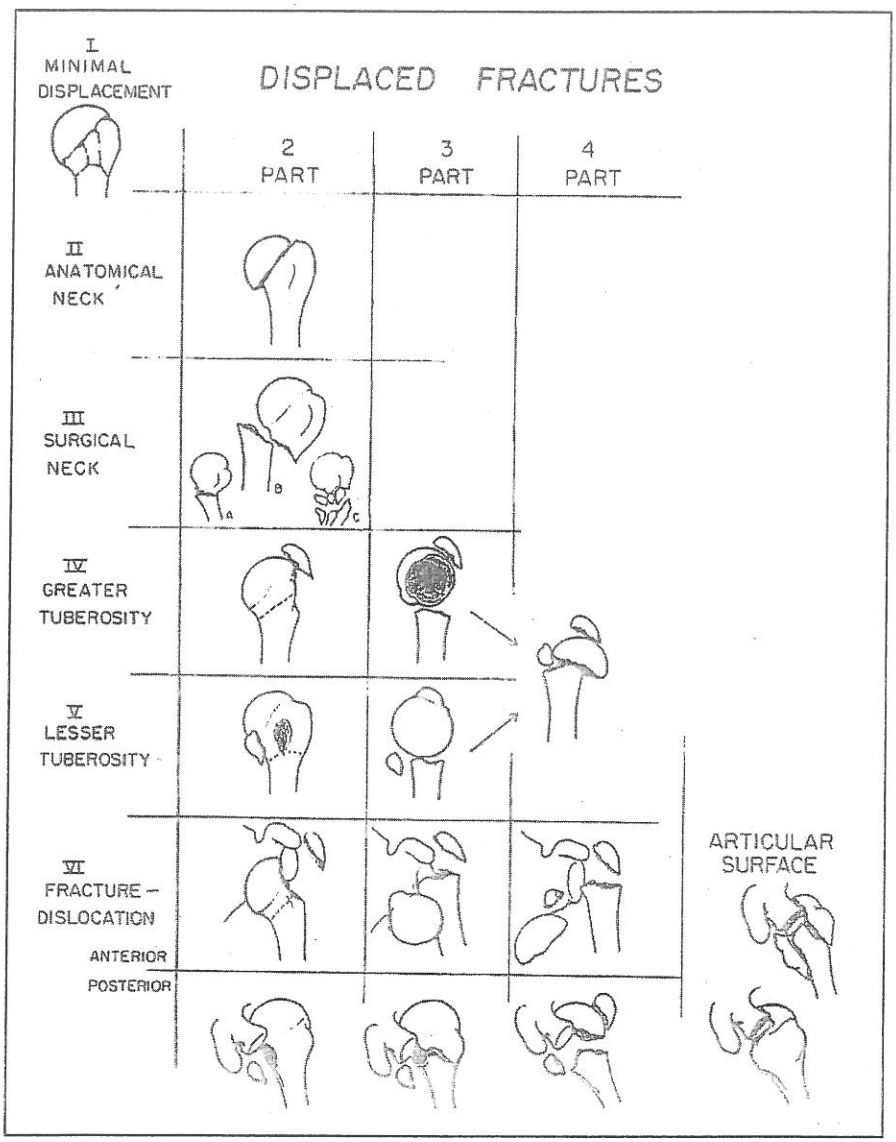
4. Rotator cuff tears
   a. The rotator cuff is made up of tendinous insertions of the following muscles that attach to the greater and lesser tuberosities of the humerus. These muscles permit abduction and control internal and external rotation of the shoulder.
      (1) Subscapularis
      (2) Supraspinatus (most frequently injured/torn)
      (3) Infraspinatus
      (4) Teres minor
   b. Mechanisms of injury
      (1) Acute tears usually occur in association with:
         (a) Forceful abduction of the arm against significant resistance (such as a fall on the outstretched arm)
         (b) A fall on the shoulder
         (c) Heavy lifting
      (2) Chronic tears (90% of all lesions) result from subacromial impingement and decreased blood supply to the tendons, both of which accompany advancing age; these tears can be insidious in onset and can occur in the absence of trauma, particularly in the elderly.
   c. Clinical presentation
      (1) Typical patient is a man ≥40 years old.
      (2) Pain over the anterior aspect of the shoulder is abrupt in onset and tearing in quality with acute tears; in chronic tears, the pain is more gradual in onset and is typically described as being worse at night.
      (3) Weak and painful abduction or, if the tear is large or complete, inability to initiate abduction; a positive "drop-arm test" (inability to hold the arm in 90° abduction) is present with significant tears.
      (4) Tenderness on palpation over the insertion site of the supraspinatus on the greater tuberosity
   d. Radiographs may be normal or show degenerative changes and, in the presence of a complete tear, may demonstrate superior displacement of the humeral head (best seen on an external rotation view).
   e. Treatment
      (1) Acute tears
         (a) Sling immobilization, analgesia with NSAIDs, and early orthopedic referral
         (b) Complete tears require early surgical repair (<3 weeks) in young, active individuals but are handled more conservatively in older, sedentary patients.
      (2) Chronic tears
         (a) Immobilize in a sling, give analgesics, and refer to an orthopedic surgeon for shoulder rehabilitation exercises and possible corticosteroid injection.
         (b) Patients who do not respond to these measures may eventually require surgical repair.

B. Scapular fractures

1. Result from high-energy trauma, particularly those involving the body or spine of the scapula
2. Because of the significant force required to produce these fractures, associated injuries are frequent and sometimes life- or limb-threatening; they occur in up to 98% of these patients and include the following:
   a. Rib fractures
   b. Ipsilateral lung injuries
      (1) Pneumothorax
      (2) Hemothorax
      (3) Pulmonary contusion
   c. Injuries to the shoulder girdle complex
      (1) Clavicle fractures
      (2) Shoulder dislocations with associated rotator cuff tears
   d. Neurovascular injuries (rare)
      (1) Brachial plexus injuries
      (2) Axillary artery or nerve injuries
      (3) Subclavian artery injury
      (4) Suprascapular nerve injury
   e. Vertebral compression fractures
3. Mechanism of injury
   a. Direct blow to the scapula
   b. Trauma to the shoulder
   c. Fall on the outstretched arm
4. Classification
   a. Based on anatomic location
   b. Fractures of the body, neck, and glenoid are most common.
5. Clinical presentation: The patient is frequently a man who was involved in a high-speed motor vehicle collision or had a significant fall. He presents with the affected arm and shoulder adducted against his body and complains of pain over the back of the shoulder. On examination, shoulder pain is increased with abduction of the arm.
6. Diagnostic evaluation
   a. Radiographs
      (1) Routine shoulder radiographs demonstrate most scapular fractures.
      (2) Axillary lateral view is helpful in evaluating fractures involving the glenoid fossa, acromion, and coracoid process.
      (3) Chest radiograph to exclude associated pulmonary injury
7. Treatment
   a. Once any associated injuries have been excluded, most scapular fractures can be managed with sling immobilization (for ≥2 weeks) and analgesia, followed by early range-of-motion exercises.
   b. Orthopedic referral for open reduction and internal fixation is usually reserved for severely displaced or angulated fractures.

C. Humeral fractures

1. Proximal humeral fractures (most commonly seen in the elderly)
   a. Mechanisms of injury
      (1) Fall on the outstretched arm (most common)
      (2) Direct blow to the lateral aspect of the arm
   b. Clinical presentation: the patient is usually an older woman with osteoporosis who presents with severe upper arm and shoulder pain after a fall; she is likely to be holding the arm in adduction.
   c. Neer classification system



Source: Neer. Displaced proximal humeral fractures. Part I. Classification and evaluation. *Journal of Bone and Joint Surgery American*. Sept 1970;52(6):1077–1089. Used with permission.

(1) An anatomic classification system for proximal humeral fractures according to the amount of displacement (fragment separation >1 cm or angulation >45°) of four segments.
   (a) Anatomic neck
   (b) Surgical neck
   (c) Greater tuberosity
   (d) Lesser tuberosity
(2) Major categories
   (a) One-part fractures (minimally displaced) demonstrate no displacement (fragment separation >1 cm or angulation >45°). These fractures account for 80%–85% of all proximal humeral fractures.
   (b) Two-part fractures demonstrate displacement of only one fragment.
   (c) Three-part fractures demonstrate the displacement of two individual fragments from the remaining proximal humerus.
   (d) Four-part fractures demonstrate displacement of all four segments.

d. Treatment
(1) In general, the amount of displacement determines how these fractures are managed. AP, lateral, and axillary (if possible) views are essential to make an accurate diagnosis.
(2) One-part fractures → immobilize with shoulder immobilizer, sling and swathe or a Velpeau dressing, analgesics, and refer for orthopedic follow-up.
(3) Two-, three-, and four-part fractures → immobilize as above and obtain emergent orthopedic referral. Many of these fractures require surgical repair. Four-part fractures may require insertion of a prosthesis.

e. Complications and associated injuries
(1) Adhesive capsulitis ("frozen shoulder") is the most common complication and can be minimized or prevented by early mobilization exercises.
(2) Associated neurovascular injuries (brachial plexus, axillary nerve, and axillary artery) should be excluded in all proximal humeral fractures, particularly surgical neck fractures (as well as fracture dislocations).
(3) Posterior shoulder dislocations frequently accompany fractures of the lesser tuberosity because of intense contraction of the subscapularis muscle, which inserts at this location; anterior and posterior dislocations occur in association with three- and four-part fractures.
(4) Avascular necrosis of the humeral head frequently complicates anatomic neck fractures, four-part fractures, and fractures of articular surfaces.

2. Humeral shaft fractures
a. Mechanisms of injury
(1) Direct blow (most common)
(2) Fall on an outstretched arm or elbow
(3) Pathologic fractures (particularly from metastatic breast cancer) are also common.

b. Usually involves middle third of the humeral shaft
c. Associated injuries
(1) Most common is damage to the radial nerve → wrist drop (inability to extend the wrist, fingers, and thumb) and loss of sensation in the first dorsal web space

(a) Nerve damage occurring at the time of injury is often due to neuropraxia and resolves spontaneously in most cases.

(b) Nerve palsy occurring after manipulation or immobilization is generally due to nerve entrapment and requires immediate surgical exploration.

(2) Ulnar and median nerve injury may also occur but are much less common.

(3) Brachial artery injury

d. Treatment

(1) Most of these fractures are managed nonoperatively with one of the following:

(a) A coaptation ("sugar-tong") splint plus sling and swathe for nondisplaced fractures

(b) A hanging cast for displaced or angulated fractures

(2) Operative management is usually reserved for patients with neurovascular compromise, soft-tissue interposition, pathologic fractures, or transverse fractures.

e. Complications

(1) Delayed union is common and may necessitate prolonged immobilization.

(2) Adhesive capsulitis (stiff/frozen shoulder) may be prevented by early initiation of circumduction exercises.

D. Elbow injuries

1. Fractures

a. Radiographic findings helpful in diagnosing occult elbow fractures that may be detected on the lateral view

(1) Fat pad signs

(a) Posterior fat pad sign: indicates distension of the joint capsule by effusion (hemarthrosis) and probable fracture; never seen on a normal elbow radiograph.

(b) Anterior fat pad sign: not as useful diagnostically as the posterior fat pad sign, because a small one is present on many normal radiographs. However, superior and anterior displacement of this fat pad suggests a probable fracture.

(2) Anterior humeral line: a line drawn along the anterior surface of the humerus and extending through the elbow that is helpful in detecting subtle supracondylar fractures; this line normally transects the middle of the capitellum but, with supracondylar extension fractures, transects the anterior third of the capitellum or passes completely anterior to it.

b. Supracondylar fractures

(1) More common in children <15 years old

(2) Two types

(a) Supracondylar extension fractures

i. Most common type

ii. Generally results from a fall on the outstretched arm with the elbow in extension or hyperextension

iii. The distal humeral fragment is displaced posteriorly, thus placing the sharp fracture fragments anteriorly, potentially injuring the brachial artery and median nerve, producing weakness of the flexor muscles of the hand and loss of two-point discrimination of the fingertips (especially the index and middle fingertips). Radial and ulnar nerve injury can also occur. Deficits that occur at the time of injury are usually neuropraxias, which resolve over time.

iv. These fractures may result in a compartment syndrome of the forearm (Volkmann ischemia) which, if not treated promptly, will result in Volkmann ischemic contracture (see also pages 509–510).

v. Treatment

- Obtain emergent orthopedic consult.
- Nondisplaced fractures should be immobilized in a posterior splint (axilla to palm) with the elbow flexed 90°. Patients may be discharged to home if close neurovascular observation and early orthopedic follow-up can be assured. Parents should be advised to return immediately if pain is unmanageable with OTC medications, or the child becomes unable to extend all fingers of the affected extremity.
- Displaced fractures require prompt reduction, followed by percutaneous pin fixation or internal fixation by an orthopedic surgeon; however, if vascular compromise threatening the viability of the extremity is present, the emergency physician may make one attempt at reducing the fracture. Patients with these fractures should be hospitalized, because delayed swelling leading to neurovascular compromise is common.

(b) Supracondylar flexion fractures

i. Usually result from a direct blow to the posterior aspect of the flexed elbow

ii. The distal humeral fragment is displaced anteriorly.

iii. These fractures are frequently open, but associated vascular injuries are uncommon.

iv. Ulnar nerve injury is the most commonly associated complication.

v. Treatment

- Nondisplaced fractures are treated with splint immobilization and early orthopedic follow-up.
- Displaced fractures require emergent orthopedic consult for reduction and percutaneous pinning; open reduction is often required for completely displaced fractures.

c. Olecranon fractures

(1) Mechanism of injury

(a) Direct blow to the olecranon

(b) Fall on the outstretched hand with the elbow in flexion

(2) Clinical presentation

(a) Swelling and tenderness over the olecranon

(b) Inability to extend the elbow against gravity or resistance due to inadequacy of the triceps mechanism

(3) Associated injuries

(a) Ulnar nerve injury (paresthesias and numbness in the ulnar nerve distribution or weakness of the interossei muscles) is common.

(b) Usually secondary to ulnar nerve contusion and resolves spontaneously over time

(4) Treatment

(a) Nondisplaced fractures → elbow immobilization in 30° flexion

(b) Fractures with >2 mm displacement → emergent orthopedic referral for surgical repair (open reduction with internal fixation)

d. Condylar fractures

(1) The distal humerus is composed of the medial and lateral condyles, each of which has an articular and nonarticular surface.

(a) Articular surfaces

i. Trochlea (on the medial condyle)

ii. Capitellum (on the lateral condyle)

(b) Nonarticular surfaces

i. Medial epicondyle

ii. Lateral epicondyle

(2) Condylar fractures usually involve both the articular surface (trochlea, capitellum) and the nonarticular surface (epicondyle) of the distal humerus.

(3) Fractures of the lateral condyle are more common than those of the medial condyle.

(4) Treatment

(a) Nondisplaced or minimally displaced fractures → immobilization in 90° elbow flexion with forearm supination (lateral condylar fracture) or pronation (medial condylar fracture) and orthopedic referral

(b) Fractures with >3 mm displacement → surgical fixation

e. Articular surface fractures (trochlea and capitellum)

(1) Mechanism of injury

(a) Trochlea and capitellum fractures usually occur in association with posterior elbow dislocations.

(b) Capitellum fractures can also result from a fall on the outstretched hand and may be associated with radial head fractures.

(2) Treatment

(a) Nondisplaced fractures → splint immobilization

(b) Displaced fractures (even if minimal) → emergent orthopedic consult and surgical repair

f. Epicondylar fractures

(1) Most commonly seen in children

(2) Medial epicondylar fractures

(a) Avulsion fractures most often occur in association with posterior elbow dislocations in children and adolescents but can also occur as a result of repeated valgus stress of the elbow in adolescents ("little leaguer's elbow"), as well as from a direct blow.

(b) Must evaluate for ulnar nerve injury (present in 60% of patients)

(c) Treatment

i. Nondisplaced (or minimally displaced) fractures → immobilization

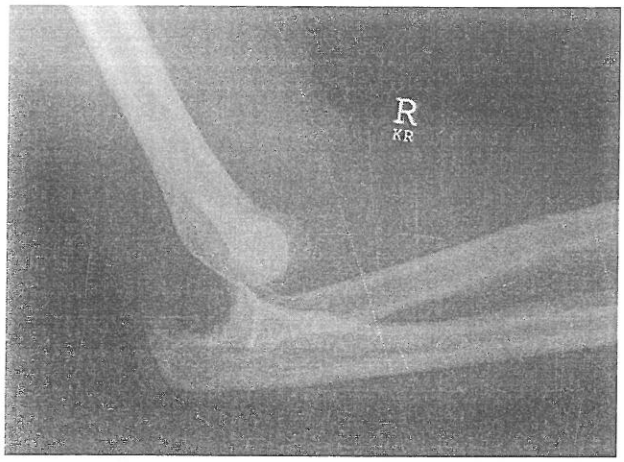
ii. Displaced fractures → orthopedic referral and surgical reduction if fragment displaced >3–5 mm or if they are intra-articular

(3) Lateral epicondylar fractures

(a) Rare, generally result from a direct blow

(b) Generally nondisplaced and treated with immobilization

2. Dislocations
   a. Posterior elbow dislocations
      (1) Most common type of elbow dislocations
      (2) Mechanism of injury: fall on the extended (or hyperextended) and abducted arm
      (3) Clinical presentation
         (a) Marked swelling with 45° flexion of the joint
         (b) Posterior prominence of the olecranon
      (4) Best demonstrated on the lateral surgical of the elbow



Source: www.lifeinthefastlane.com. Used with permission.

      (5) Associated injuries
         (a) Fractures about the elbow (30%–60% of cases)
         (b) Injuries to the ulnar and median nerves (common)
         (c) Brachial artery injury (occurs in up to 8% of cases; should be suspect when a large opening between the tip of the olecranon and the distal humerus is palpated on examination or seen on radiograph)
      (6) Treatment is reduction. Orthopedic consultation is usually not necessary to proceed, and reduction should be done immediately if there is neurovascular compromise. Neurovascular status should be assessed before and after reduction.
         (a) Provide procedural sedation.
         (b) Apply traction distally at the wrist, while an assistant immobilizes the humerus.
         (c) While maintaining traction at the wrist, flex the elbow and apply posterior pressure to the distal humerus.
         (d) A "clunk" will be heard or felt as the elbow is reduced and the articular surfaces mesh.
         (e) Move the elbow through its full range of motion to check stability, and then reassess the neurovascular status.
         (f) Immobilize the elbow in a long-arm posterior splint in 120° (full flexion) or as much flexion as circulation permits and obtain radiographs (after reduction).
         (g) Patients can be discharged with instructions to apply ice, elevate, and watch for signs of vascular compromise. If the patient is not reliable, he or she should be admitted and observed for possible compartment syndrome.
   b. Anterior elbow dislocations (uncommon)
      (1) Mechanism of injury: a blow to the olecranon with the elbow in flexion

(2) Clinical presentation: the forearm is elongated and supinated while the elbow is generally held in full extension

(3) Associated injuries

(a) Incidence of vascular impairment is much higher than with posterior dislocations.

(b) Associated avulsion of the triceps mechanism is common.

(4) Treatment

(a) Once the neurovascular status has been assessed and procedural sedation provided, an assistant immobilizes the humerus while the physician applies in-line traction to the wrist (with one hand) and downward and backward pressure to the proximal forearm (with the other hand).

(b) Elbow dislocations may have associated fractures that may block reduction or render it unstable. Because of this (and the potential for significant neurovascular sequelae), consultation with an orthopedic surgeon should be obtained (if possible) before any reduction attempts of a fracture dislocation.

c. A severe disruption of the elbow joint with any type of dislocation may be accompanied by injury to the brachial artery and should be suspected when a wide opening (olecranon to distal humerus) is palpated or seen on a lateral radiograph.

d. Radial head subluxation (nursemaid elbow)

(1) Subluxation of the radial head without associated ulnar fracture occurs with some regularity in children <5 years old.

(2) Mechanism of injury: abrupt longitudinal traction on the hand or forearm with the arm in pronation pulls the annular ligament over the radial head (annulus).

(3) Clinical presentation

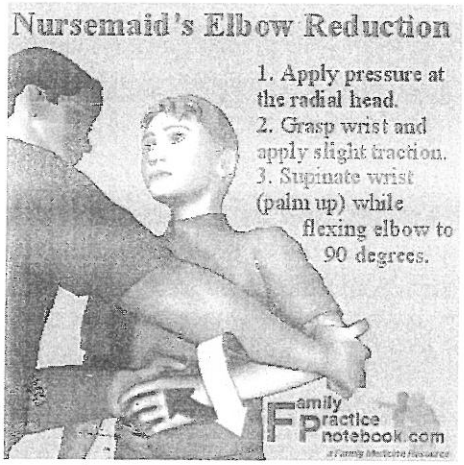
(a) The child presents with the arm dangling at his or her side, unwilling to move it.

(b) The elbow is flexed, and the arm is held in passive pronation.

(c) There is resistance to and pain with full supination and with direct palpation over the radial head.

(4) Radiographs (if obtained) are usually normal. They are only helpful in excluding other diagnoses and should be ordered only when the history is unclear or reduction is unsuccessful.

(5) Reduction can be accomplished via the supination method by placing the thumb on one hand on the radial head and using the other hand to supinate the forearm and flex the elbow. A click is often palpated over the radial head as it reduces.



Source: www.fpnotebook.com (http://www.fpnotebook.com/_media/ElbowNursemaidsReduction.jpg). Used with permission.

(6) **A second technique, the hyperpronation method, has a higher success rate and may be associated with less pain. In this method, the examiner applies pressure over the radial head while hyperpronating the wrist. The elbow is then flexed if reduction has not already occurred.**

E. Radial and ulnar injuries

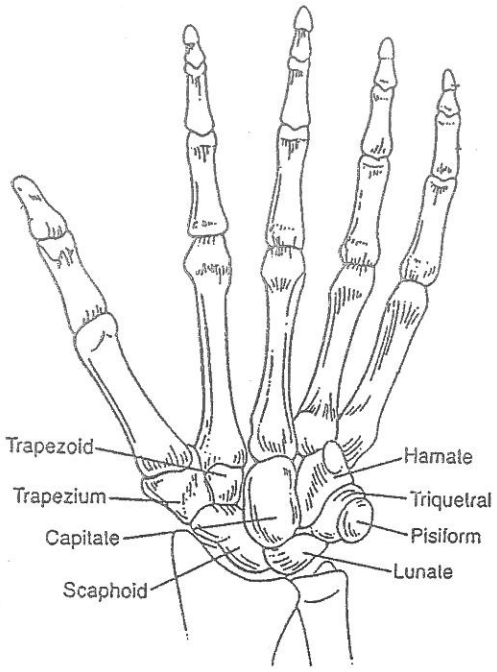
1. Radial head fracture
   a. Often results from a fall on the outstretched hand
   b. Clinical presentation
      (1) Physical examination reveals tenderness and swelling over the radial head; pain is increased with forearm supination.
      (2) Also check for distal radioulnar dissociation (Essex-Lopresti lesion).
   c. Diagnostic evaluation: although nondisplaced fractures may not be visible on initial radiographic evaluation, a bulging anterior fat pad sign (sail sign) or a posterior fat pad sign is usually present, suggesting the diagnosis.
   d. Associated injury to the articular surface of the capitellum is common.
   e. Treatment
      (1) Nondisplaced fractures are treated with sling immobilization followed by early range of motion exercises as tolerated. If the swelling is very painful, the joint may be aspirated through the posterolateral triangle, but this is rarely necessary.
      (2) Comminuted and displaced fractures are treated with immobilization in a long-arm posterior splint and early (2–5 days) orthopedic referral for screw fixation or radial head excision (with or without silastic implant) if one of the following exists:
         (a) Marked comminution of the fracture
         (b) Angulation of the articular surface that is >30°
         (c) >2 mm offset in a two-part fracture
         (d) Fracture involving more than one-third of the articular surface
2. Galeazzi fracture
   a. A fracture of the distal radial shaft associated with a distal radioulnar dislocation (radiographic signs may be subtle)
   b. Mechanisms of injury
      (1) Direct blow to the back of the wrist
      (2) Fall on the outstretched hand in forced pronation
   c. Treatment: open reduction and internal fixation
3. Nightstick fracture
   a. An isolated fracture of the shaft of the ulna
   b. Mechanism of injury: a direct blow to the subcutaneous border of the ulna that usually occurs when a patient raises up the forearm to protect his or her face from a blow
   c. Treatment
      (1) Nondisplaced fractures: immobilization in a long-arm cast
      (2) Displaced fractures (those with >10° angulation or displacement >50% of the diameter of the ulna): orthopedic referral for open reduction and internal fixation

4. Monteggia fracture
   a. A fracture of the proximal third of the ulna combined with dislocation of the radial head (usually anterior)
   b. Mechanisms of injury
      (1) Direct blow to the posterior aspect of the ulna
      (2) Fall on the outstretched hand with forearm in forced pronation
   c. Diagnostic evaluation
      (1) The ulnar fracture is often apparent on AP and lateral views of the forearm, but the radial head dislocation is missed in as many as 25% of cases.
      (2) This can be avoided by remembering to assess the alignment of the radial head with the capitellum (radiocapitellar line on lateral radiograph); if the radial head is in its normal anatomic position, a line drawn through the radial shaft and head should intersect the capitellum in all views.
   d. Associated injury to the radial nerve is common; usually resolves spontaneously in 6–8 weeks.
   e. Treatment
      (1) In adults, open reduction and internal fixation of the ulnar fracture followed by closed reduction of the radial head dislocation is the preferred treatment and is associated with the best functional outcome.
      (2) Children can usually be treated with closed reduction under general anesthesia.
5. Fractures of both the radius and ulna
   a. Usually displaced
   b. Mechanism of injury: most commonly a direct blow to the forearm
   c. Associated injuries
      (1) Peripheral nerve deficits (radial, ulnar, and median) occur infrequently with closed injuries but can be seen with open fractures.
      (2) Development of compartment syndrome is a major concern.
   e. Treatment
      (1) Nondisplaced fractures (rare): immobilization in a sugar tong splint
      (2) Displaced fractures: generally open reduction and internal fixation with compression plates is required (in adults); children can sometimes be treated with closed reduction.
   f. Complications: compartment syndromes (both anterior and posterior), malunion, and nonunion

F. Hand and wrist injuries

1. Essential anatomy

a. The eight carpal bones



b. The ulnar nerve

(1) Runs deep to the flexor carpi ulnaris tendon and through Guyon canal

(2) Innervates the following muscles:

(a) Hypothenar eminence

(b) Interosseous muscles

(c) Lumbrical muscles of the ring and little fingers

(d) Adductor pollicis brevis

(e) In the forearm, the flexor carpi ulnaris and ulnar aspect of the flexor digitorum profundus

(3) Provides sensation to palmar and dorsal aspects of the ulnar side of the hand, the little finger, and the ulnar half of the ring finger

(4) Testing

(a) Motor test of normal function → abduction of fingers against resistance (patient can spread fingers apart)

(b) Sensation → best tested over the volar tip of the little finger

(5) Loss of ulnar nerve function → inability to hold a piece of paper between fingers (early) or claw hand (late)

c. The median nerve

(1) Runs through the carpal tunnel between the flexor carpi radialis and the palmaris longus

(2) Innervates the following muscles:

(a) Pronator teres

(b) Flexor carpi radialis

(c) Flexor digitorum superficialis and profundus (radial part)

(d) Flexor pollicis longus
(e) Pronator quadratus

(3) The thenar motor branch supplies the following:
(a) Abductor pollicis brevis
(b) Opponens pollicis
(c) Flexor pollicis brevis

(4) The common digital branches innervate the lumbrical muscles of the index and middle fingers

(5) Provides sensation to the palm on the radial side of the hand and the palmar aspect of the radial three and one-half fingers, as well as the dorsal aspect of the tips of the index and middle fingers and the radial half of the ring finger

(6) Testing
(a) Motor test of normal function → opposition of the thumb to each finger while watching thenar muscles for contractions
(b) Sensation → best tested over the volar tip of the index finger

(7) Loss of median nerve function → carpal tunnel syndrome; with thenar atrophy in the late stages → monkey hand

d. The radial nerve

(1) Innervates the following muscles:
(a) Triceps
(b) Brachioradialis
(c) Extensor carpi radialis longus
(d) Extensor carpi radialis brevis
(e) Supinator
(f) Extensor digitorum communis
(g) Extensor digiti minimi
(h) Extensor carpi ulnaris
(i) Abductor pollicis longus
(j) Extensor pollicis brevis
(k) Extensor pollicis longus
(l) Extensor indicus proprium

(2) Does not innervate any of the intrinsic muscles of the hand

(3) Provides sensation to the dorsum of the radial aspect of the hand, the dorsum of the thumb, the dorsal aspect of the index and middle fingers, and the radial half of the ring finger as far distally as the proximal interphalangeal joints

(4) Testing
(a) Motor test of normal function → extension of wrist and fingers against resistance
(b) Sensation → best tested over the dorsal web space between the thumb and index finger

(5) Primary function → extension of the wrist and metacarpophalangeal joints

(6) Loss of radial nerve function → wrist drop, loss of finger extension

e. The intrinsic muscles of the hand
   (1) Thenar
   (2) Hypothenar
   (3) Abductor pollicis
   (4) Lumbricals
   (5) Interossei

f. The six groups of extrinsic extensor muscles
   (1) Abductor pollicis longus and extensor pollicis brevis
   (2) Extensor carpi radialis longus and brevis tendons
   (3) Extensor pollicis longus tendon
   (4) Extensor indices proprius and extensor digitorum communis
   (5) Extensor digiti minimi
   (6) Extensor carpi ulnaris

g. The extrinsic flexor muscles
   (1) Flexor pollicis longus
   (2) Flexor digitorum superficialis
   (3) Flexor digitorum profundus
   (4) Flexor carpi ulnaris
   (5) Flexor carpi radialis
   (6) Palmaris longus

2. Regional nerve blocks can be particularly useful when treating finger and hand injuries.
   a. Digital blocks
      (1) Local infiltration into the restricted space of the finger is very painful and can impair capillary refill; digital blocks are, therefore, better than local infiltration for most finger injuries.
      (2) Sensation to the finger (dorsal and volar surfaces and the interphalangeal joints) is provided by the palmar and dorsal digital nerves, which run along the lateral aspects of each phalanx.
      (3) Digital blocks are performed by using one of three approaches (each of which has advantages and disadvantages).
         (a) Dorsal approach: needle is directed into the dorsum of the hand at the metacarpals.
         (b) Palmar approach: needle is directed into the palm over the metacarpal head (very painful).
         (c) Web space approach: needle is directed into the interdigital web space.
   b. Nerve blocks (wrist)
      (1) Useful for extensive hand injuries (particularly the palm, which is very painful to inject into) and metacarpophalangeal joint injuries, but they require more time and training to learn and, therefore, are less commonly used.
      (2) Sensation to the hand is provided by the median, ulnar, and radial nerves; therefore, if complete anesthesia of the hand is needed, all three of these nerves must be blocked (requires multiple injections).
      (3) These blocks are performed on the volar aspect of the wrist at the proximal skin crease where the tendons are easily palpated and provide landmarks to guide the injections.

(4) More detailed information on performing these blocks can be found in *Roberts and Hedges Clinical Procedures in Emergency Medicine,* 5th ed, pages 517–528.

3. **Carpal injuries**
   a. **Scaphoid (navicular) fracture**
      (1) **Most common carpal fracture**
      (2) **Mechanism of injury: fall on the outstretched hand**
      (3) **Initial AP, lateral, and scaphoid radiographic views of the wrist may not demonstrate a fracture in ≥10% of cases. Repeat radiographs of the wrist in 2 weeks will often demonstrate the fracture line.**
      (4) **Clinical presentation**
         (a) **Tenderness in the region of the anatomic snuff box**
         (b) **Pain referred to the anatomic snuff box with longitudinal compression of the thumb (axial loading) or with supination of the hand against resistance; if these findings are present, treat as a fracture.**
      (5) **Treatment**
         (a) **Nondisplaced and clinically suspected fracture: immobilization in a thumb spica splint and referral to an orthopedist/hand surgeon**
         (b) **Displaced fractures usually require open reduction and fixation.**
      (6) **Complications**
         (a) **Avascular necrosis of the proximal fragment; the more proximal, oblique, or displaced the fracture, the greater the risk because the vascular supply enters the distal part of the bone.**
         (b) **Delayed union, malunion, and nonunion**
   b. Triquetrum dorsal chip fracture
      (1) Second most common carpal fracture
      (2) Mechanisms of injury
         (a) Fall on the outstretched hand
         (b) Direct blow to the dorsum of the hand
      (3) Clinical presentation: tenderness immediately distal to the ulnar styloid on the dorsal aspect of the wrist
      (4) Best visualized on the lateral view of the wrist
      (5) Treatment: immobilization in a volar splint
   c. Lunate fracture
      (1) Third most common carpal fracture
      (2) Mechanism of injury: fall on the outstretched hand
      (3) Clinical presentation: pain and tenderness over the mid-dorsum of the wrist that is increased by axial compression of the third metacarpal
      (4) Plain radiographs of the wrist are often normal; therefore, treatment should be started on clinical grounds alone.
      (5) Treatment: immobilization in a thumb spica splint and orthopedic referral
      (6) Complications
         (a) Avascular necrosis of the proximal segment (Kienbock disease) is a serious complication.
         (b) It is seen most often in patients with congenital shortening of the lunate, but it also results from inadequate immobilization of a lunate fracture.

d. Dislocations
   (1) Mechanism of injury: violent hyperextension
   (2) Refer all to a hand surgeon
   (3) Lunate dislocation
      (a) The lunate may dislocate either volarly (most common) or dorsally.



Source: James Heilman, MD
(Wikimedia: http://en.wikipedia.org/wiki/File:LunatedislocationL.jpg)

      (b) Clinical presentation
         i. There is pain, swelling, and marked loss of flexion with the wrist, hand, and arm held in anatomic position.
         ii. Occasionally, the patient complains of tingling in the three radial digits (acute carpal tunnel syndrome).
      (c) Findings on wrist radiograph
         i. The diagnosis may be suggested on the AP view: the lunate is normally squarish in appearance but when it dislocates, it becomes triangular in shape ("piece of pie" sign). There is also foreshortening of the wrist and loss of the normal space between the capitate and the lunate.
         ii. The diagnosis is confirmed by a good lateral view that demonstrates displacement of the lunate volarly relative to the capitate and carpus, which remain in their normal alignment ("spilled teacup" sign).
      (d) Associated scaphoid injuries are common.
   (4) Perilunate dislocation (most common wrist dislocation)
      (a) May be associated with a fracture or dislocation of the scaphoid
      (b) The lunate remains in anatomic position relative to the forearm while the capitate is displaced dorsally because of disarticulation of the capitolunate joint.
      (c) Diagnostic evaluation: as with a lunate dislocation, a perilunate dislocation is best diagnosed by a true lateral film of the wrist.

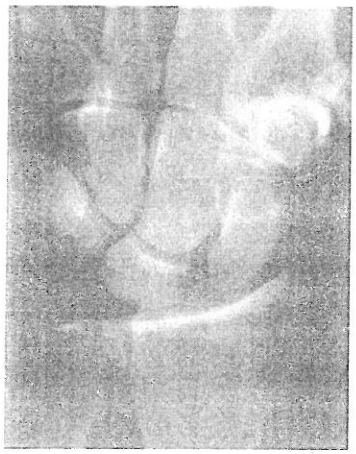


Source: Hellerhoff
(Wikimedia: http://en.wikipedia.org/wiki/File:Perilunaere_Luxation.jpg)

(5) Scapholunate dislocation

(a) Clinical presentation: pain with wrist hyperextension and a snapping sensation when the wrist is deviated in either the radial or ulnar direction

(b) Diagnostic evaluation: radiologic signs



Source: Wikimanos
(Wikimedia: http://en.wikipedia.org/wiki/File:Dynamic_S-L-grip.jpg)

i. AP film: The scaphoid is foreshortened and has a dense ring-shaped image around its distal edge ("signet ring" sign). There is a widening >3 mm in the space between the lunate and scaphoid ("Terry Thomas" sign).

ii. Lateral film: the angle between the scaphoid and the lunate is increased (>60°).

(c) Treatment: place the arm in a radial gutter splint or posterior mold for orthopedic referral.

4. Metacarpal injuries

a. Metacarpal neck fractures

(1) Result from a punch with a clenched fist

(2) Almost all are unstable.

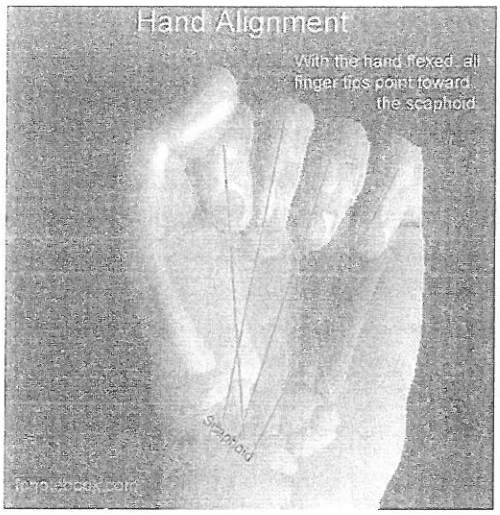
(3) The proximal fragment angulates in the dorsal direction, while the distal fragment angulates in the volar direction.

(4) The amount of angulation that is acceptable varies directly with the normal mobility of the involved metacarpal; the greater the mobility of the metacarpal, the greater the degree of angulation that can be tolerated.

(a) Metacarpal neck fractures of the ring and little fingers ("Boxer's fracture"); up to 20° and 40° of angulation, respectively, is acceptable.

(b) Metacarpal neck fracture of the index and long fingers; ≤15° of angulation is acceptable.

(5) Rotational deformities, if present, must be completely corrected; look for malalignment of the plane of the fingernails when the fingers are viewed in the partially flexed position.



Source: www.fpnotebook.com (http://www.fpnotebook.com/_media/OrthoHandPositionFistToScaphoid.jpg). Used with permission.

b. Metacarpophalangeal joint dislocations

(1) Metacarpophalangeal dislocations of fingers are rare, while those of the thumb are relatively common.

(2) Usually dorsal and result from hyperextension forces

(3) Types

(a) Simple = subluxation

(b) Complex = complete dislocation

(4) Radiographs

(a) Should be taken both before and after reduction to exclude associated fractures and confirm adequate reduction

(b) The lateral view usually demonstrates an obvious dislocation.

(5) Treatment

(a) Simple dislocations can usually be managed with closed reduction with adequate anesthesia (requires a wrist block of the ulna, median, and/or radial nerves, depending on which finger is involved), splinting in flexion, and referral to a hand surgeon.

(b) Complex dislocations often cannot be reduced by closed reduction because of interposition of the volar plate in the metacarpophalangeal joint or entrapment of the metacarpal head between the lumbrical tendon and a flexor tendon. Irreducible metacarpophalangeal joint dislocations should be splinted and referred to a hand surgeon for open reduction and operative repair.

(6) Complications

(a) Volar plate injuries

(b) Bony avulsions

(c) Thickening and stiffness of the joint

5. Bennett fracture/subluxation: an unstable fracture at the base of the first metacarpal, often caused by punching; it should be suspected when evaluating a "sprained thumb."
6. Gamekeeper's or skier's thumb (torn ulnar collateral ligament)
   a. Mechanism of injury
      (1) Acute and forceful radial deviation of the thumb
      (2) Commonly occurs in skiing accidents when the ski pole forcefully abducts the thumb at the metacarpophalangeal joint as the hand hits the ground in a fall
   b. Clinical presentation
      (1) Tenderness along the ulnar aspect of the thumb, which is most exquisite at the metacarpophalangeal joint
      (2) Thumb grasp and pinch are weak.
   c. Diagnostic evaluation
      (1) Obtain radiographs of the thumb to exclude associated fractures (present in up to 30% of cases).
      (2) Assess joint stability.
         (a) Apply a lateral stress (radial abduction) to the metacarpophalangeal joint of the injured thumb (after adequate anesthesia). You must learn how to do this; failure to diagnose this injury may result in chronic instability of the thumb and debilitating osteoarthritis.
         (b) Stress the normal thumb in the same manner.
         (c) The presence of 10°–20° of laxity in the injured thumb when compared with the normal thumb is consistent with a complete tear and requires surgical repair.
   d. Treatment
      (1) Incomplete tear (some stability = <10° laxity): thumb spica splint and follow-up with a hand surgeon.
      (2) Complete tear (no stability = 10°–20° laxity): surgical repair
7. Distal forearm fractures
   a. Colles' fracture
      (1) Transverse fracture of the metaphysis of the distal radius with dorsal displacement of the distal fragment
      (2) Mechanism of injury: a fall on the outstretched hand
      (3) Clinical presentation: classic "dinner fork" deformity of the wrist produced by dorsal displacement of the fracture

(4) Associated injuries
(a) Ulnar styloid fracture (present in 60% of cases)
(b) Median nerve injury
(5) Treatment
(a) Nondisplaced fractures: immobilize in a coaptation (sugartong) splint, and refer to an orthopedic surgeon for follow-up.
(b) Displaced fractures: require prompt reduction, usually accomplished with traction (using finger traps) and manipulation; follow with a coaptation (sugar-tong) splint and orthopedic referral. Adequate local anesthesia can be achieved by injecting 5–10 mL of lidocaine directly into the hematoma of the fracture at the fracture site.
i. Immobilization should ultimately be in slight flexion and ulnar deviation to maintain/restore the normal length and tilt of the radius.
ii. If a cast is applied, it should be split (bivalved) before the patient is discharged.

b. Smith fracture
(1) Transverse fracture of the distal radial metaphysis with volar displacement of the distal fragment
(2) Mechanisms of injury
(a) Fall on the outstretched hand with the forearm in supination
(b) Direct blow to the dorsum of the distal radius or wrist with the hand flexed and the forearm pronated
(3) Associated injury: median nerve should be excluded.
(4) Treatment
(a) After adequate anesthesia (hematoma block), these fractures are reduced with traction (using finger traps) and manipulation.
(b) Follow with immobilization in a sugar-tong splint and referral for orthopedic follow-up.

8. Tendon injuries
a. General principles of evaluation
(1) First, observe the resting posture of the hand; any change in the normal flexion cascade is suspicious of a tendon injury.
(2) Second, examine the wound with the hand/finger in the position it was in at the time of the injury; this will aid in determining the location of the tendon injury relative to the skin laceration (if present).
(3) Finally, evaluate the wound/tendon as the hand/finger is taken through its full range of motion.

b. Flexor tendon injuries should be repaired by a hand surgeon. These tendons are most often injured in association with lacerations, although closed traumatic disruption can also occur. A common example of the latter is the "jersey finger," in which the flexor digitorum profundus tendon is avulsed when one football player grabs the jersey of another and his finger gets caught. Examination of the flexor tendons is as follows:
(1) Flexor digitorum profundus tendon: immobilize the proximal interphalangeal and metacarpophalangeal joints in extension and ask the patient to flex the tip of the finger; inability to flex the distal interphalangeal (DIP) joint indicates a profundus tear that is usually associated with a volar plate slip at the proximal interphalangeal (PIP) joint.

(2) Flexor digitorum superficialis tendon: hold the uninjured fingers in extension and ask the patient to flex the injured finger; this blocks the action of the profundus tendon and allows an isolated test of the superficialis tendon.

(3) It is not adequate merely to test the function of these tendons, because patients with 90% full-thickness lacerations still have normal (although painful) range of motion. These partial tears are detected by evaluating the strength of these tendons against resistance; patients with partial tears will have weakness against resistance. Direct visualization of the tendon through the entire range of motion is crucial. Treatment of partial tendon lacerations is controversial and should be determined in consultation with a hand surgeon. Many are treated with protective splinting alone.

c. Extensor tendon injuries are usually closed. (Be able to identify on pictorial.)

(1) Mallet finger

(a) Extensor tendon laceration or disruption at the DIP joint (may or may not be associated with avulsion chip fracture)

(b) Clinical presentation: The patient is unable to extend the DIP joint actively.

(c) Mechanism of injury: usually a blow to the tip of the extended finger producing sudden forced flexion

(d) Treatment

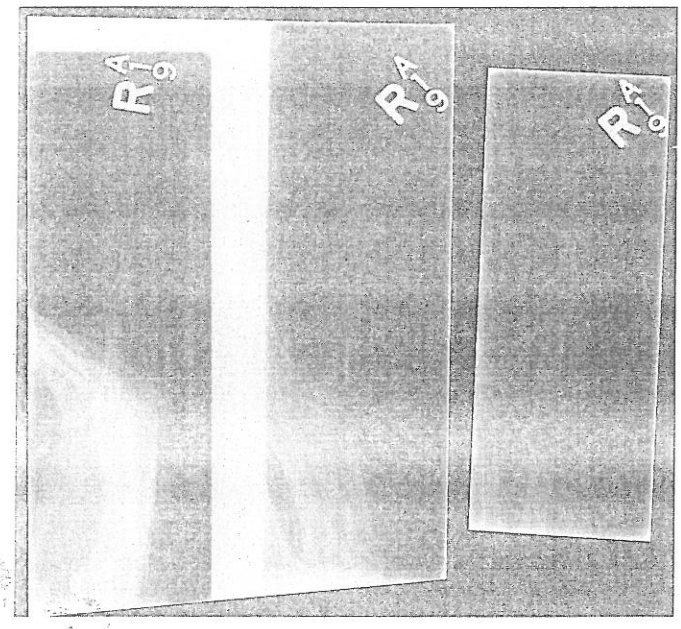
i. If there is no associated fracture → splint the DIP in extension to slight hyperextension for 6–8 weeks.

ii. If there is an associated fracture → the treatment is either splinting as above or surgical pinning of the avulsed fragment using Kirschner wire fixation.

(e) Complications

i. A delayed complication of an untreated mallet finger is the "swan neck deformity," in which there is hyperextension of the PIP joint in addition to the mallet flexion deformity of the DIP joint.

ii. It is the result of increased extension forces on the PIP joint and is produced by proximal and dorsal migration of the lateral bands.



Courtesy of Michael C. Bond, MD

(2) Boutonniere deformity
(a) Rupture of the central slip of the extensor tendon at the PIP.
(b) Clinical presentation: characterized by flexion of the PIP joint and hyperextension of the DIP joint
(c) Mechanism of injury: a direct blow to (or laceration of) the PIP joint or forced flexion of the PIP joint against resistance.
(d) This deformity is not always present immediately after the injury, but rather often develops over time (1–2 weeks after the injury) because the lateral bands of the extensor tendon slip volar to the axis of the PIP joint and so become paradoxical flexors of this joint.
(e) Treatment: splint the PIP in extension and then refer to a hand surgeon for possible operative repair.

d. De Quervain stenosing tenosynovitis
(1) An inflammation of the extensor tendons of the thumb
(a) Abductor pollicis longus
(b) Extensor pollicis brevis
(2) Clinical presentation
(a) Complaint of pain along the radial aspect of the wrist that is exacerbated with use of the thumb
(b) Palpation reveals tenderness over the radial styloid.
(c) A positive Finkelstein test (pain on ulnar deviation of the wrist while the thumb is flexed and held in the palm by the other fingers) confirms the diagnosis.
(3) Mechanism of injury: overuse of the thumb
(4) Treatment
(a) Administer oral NSAIDs.
(b) Place in thumb spica splint.
(c) Inject the first dorsal compartment with a bupivacaine and triamcinolone combination; this can be done on the initial evaluation or reserved for those patients in whom conservative therapy has not been effective. (Repeated injections are associated with a risk of subsequent tendon rupture.)

9. Amputated digits
a. Preservation of the amputated part
(1) After irrigating the amputated part with normal saline to remove gross contamination, wrap it in sterile gauze moistened with lactated Ringer's or normal saline and place it in a sterile, watertight container.
(2) Store the container in ice water.
b. Criteria for reimplantation
(1) A young, stable patient
(2) A sharply incised wound with minimal associated damage
(3) An amputated thumb
(4) Multiple-digit amputation
(5) Hand or forearm amputation
(6) Amputation in a child

c. **Contraindications to reimplantation**
   (1) **Absolute**
      (a) **Unstable patient with other life-threatening injuries**
      (b) **Severe crush injury**
   (2) **Relative**
      (a) **Severely damaged or contaminated part**
      (b) **Single-digit amputation (other than the thumb)**
      (c) **Avulsion injury**
      (d) **Serious underlying systemic illness (eg, CHF, diabetes mellitus)**
      (e) **Prolonged warm ischemia (≥12 hours)**
      (f) **Prior injury/surgery to affected part**
      (g) **Emotionally unstable patient**

III. PELVIS AND HIP INJURIES

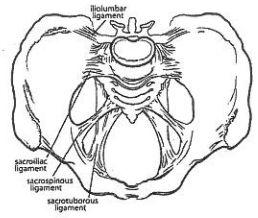
A. Essential anatomy of the pelvis

1. Pelvic bones: innominate (consists of the ilium, ischium, and pubis), sacrum, and coccyx
2. On the AP view of the pelvis, the symphysis pubis is usually 5 mm wide, and the sacroiliac joint is normally 2–4 mm wide.
3. The lumbar and sacral nerves run through the pelvis.
4. The ileopectalline divides the upper (false) pelvis and the lower (true) pelvis. Injuries to the true pelvis may be associated with injuries to the following structures:
   a. Bladder or urethra
   b. Descending colon
   c. Sigmoid colon
   d. Rectum
   e. Anus
   f. Nerve roots
   g. Vasculature

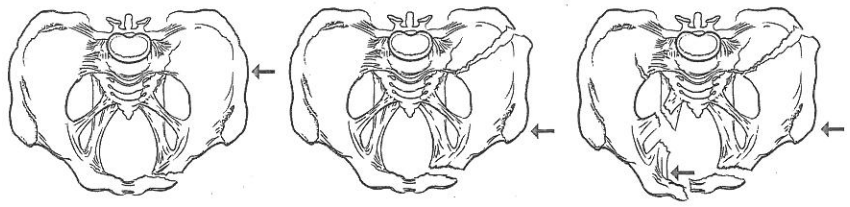
B. Pelvic fractures

1. Mechanism of injury
   a. Motor vehicle and motorcycle collisions (most common cause of injury in adults)
   b. Falls
   c. Crush injuries
   d. Pedestrian hit by motor vehicle (leading cause of injury in children)
2. **Classification: Several different systems have developed over the years to classify pelvic fractures. The central focus of most of these systems is the underlying stability of the pelvis. The Young-Burgess system is most favored by emergency physicians, because it is valuable in predicting hemorrhage related to pelvic fracture and local vascular injury. This system is based on the mechanism of injury (AP compression or lateral compression) as well as the degree of pelvic instability.**
3. **Classification types: see page 472 for illustrations; arrow indicates direction of force.**

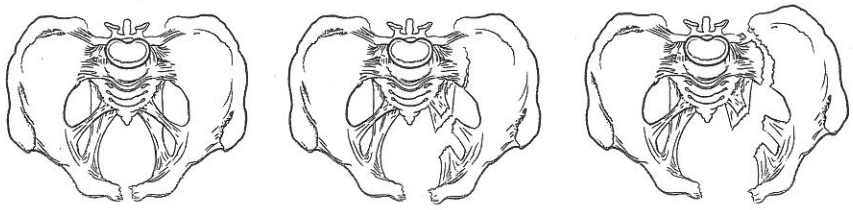
4. Lateral compression
   a. Type I: posteriorly directed force → sacral crushing and horizontal pubic ramus fractures ipsilaterally
   b. Type II: anteriorly directed force → horizontal pubic ramus fractures with anterior sacral crushing and disruption of the posterior sacroiliac joints or fractures through the iliac wing
   c. Type III: anteriorly directed force → continued, causing external rotation of the contralateral side
      (1) Sacroiliac joint opened posteriorly
      (2) Sacrotuberous and spinous ligaments disrupted
5. Anteroposterior (AP) compression
   a. Type I: symphysis disrupted but posterior ligamentous structures intact
   b. Type II: continues type I, with disruption of the sacrospinous and possibly the sacrotuberous ligaments and an anterior sacroiliac joint opening
   c. Type III: continued force disrupts the sacroiliac ligaments
6. Vertical shear
   a. Vertical fractures in the rami; disruption of all posterior ligaments
   b. Equivalent to an AP type III or completely unstable and rotationally unstable fracture



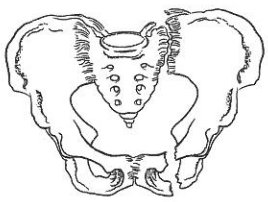
Ligamentous anatomy of the pelvis



A, lateral compression I II III



B, anteroposterior (AP) compression I II III

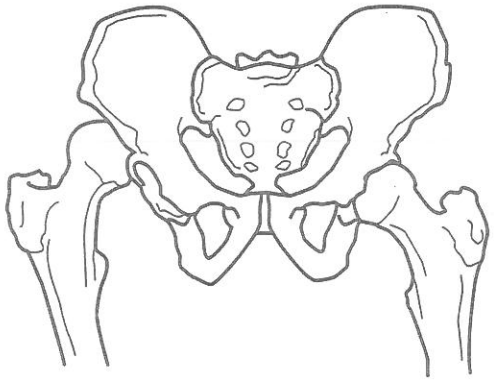


C, vertical shear

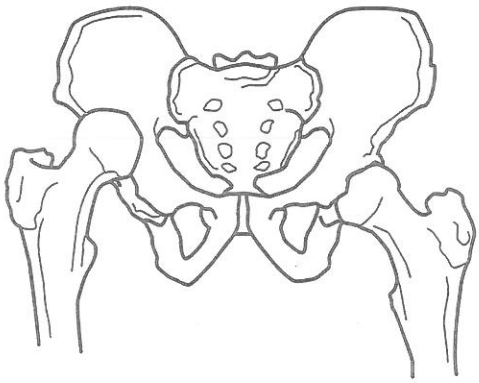
7. Clinical presentation
   a. Destot sign: a superficial hematoma above the inguinal ligament or on the scrotum
   b. Earle sign: on rectal examination, a large hematoma or tenderness along the fracture line or palpation of a bony prominence
   c. Roux sign: a diminished distance measured from the greater trochanter to the pubic spine on one side compared with the other (indicates an overlapping fracture of the anterior pelvic ring)
8. Diagnostic evaluation: CT
   a. Superior to standard radiographics in evaluating the acetabulum, posterior (femorosacral) arch, and sacroiliac joint
   b. Often provides information on the degree of pelvic instability and the presence of a retroperitoneal hematoma
9. Associated injuries: because the developing pelvis provides a lesser degree of protection, children have a higher rate of concomitant injuries than adults.
   a. Hemorrhage
      (1) The primary cause of death in patients with pelvic fractures; up to 6 L of blood can be accommodated in the retroperitoneal space.
      (2) Most common in patients with Type III and posterior fractures
      (3) Up to 50% of patients require transfusion.
      (4) May initially be controlled by application of a pelvic binder or tying a sheet across the pelvis; external fixation and angiographic embolization are also useful in selected patients and may be subsequently required.
   b. Injuries of the urethra and bladder
      (1) Most commonly associated pelvic injuries
      (2) Urethral injuries (particularly posterior urethral tears in men) are more common than bladder injuries.
   c. Vaginal laceration or rupture (relatively uncommon but may be seen with anterior pelvic fractures)
   d. Nerve damage (most commonly occurs in association with posterior pelvic fractures)
   e. Ruptured diaphragm (not uncommon and is often missed)
   f. Rectal injuries
      (1) Uncommon
      (2) Generally occur in association with ischial and urinary tract injuries
      (3) Prophylactic antibiotics should be administered without delay.
   g. Thoracic aortic rupture (traumatic aortic disruption): incidence is 8 times greater in patients with pelvic fractures than in patients with blunt abdominal trauma.
10. Complications of pelvic fractures
   a. Sepsis
   b. Thromboembolic complications (pulmonary and fat emboli)
   c. Malunion or delayed union
   d. Chronic pain

11. Specific pelvic fractures
    a. Pubic rami
        (1) A single pubic ramus fracture is the most common pelvic fracture. A fall on the buttocks may be missed (or not visible) on plain radiograph; order an MRI if in doubt.
        (2) A "straddle injury" is a fracture of all four pubic rami, such as occurs in a fall on the crotch with the legs apart.
    b. Iliac crest (result from impact collisions)
        (1) Duverney fracture (pelvic wing fracture)
        (2) Ilium fracture → pelvic ring disruption, visceral injuries
    c. Multiple pelvic fractures (Malgaigne fracture)
        (1) Involves the pubic rami bilaterally and the ilium or sacrum
        (2) Results from vertical shear forces (eg, fall from a height)

C. Hip dislocations (an orthopedic emergency [in the absence of a prosthetic femoral head])
1. Anterior: 10% of hip dislocations (Be able to identify on pictorial.)
    a. Classified according to the final resting position of the femoral head
        (1) Superior iliac: femoral head is palpable in the area of the superior iliac spine.
        (2) Superior pubic: femoral head rests near the pubis.
        (3) Inferior (obturator): femoral head lies in the obturator foramen.
    b. Mechanism of injury
        (1) Extreme abduction causes the femoral head to be pushed out through a tear in the anterior capsule.
        (2) Causes include:
            (a) Motor vehicle collision
            (b) A fall
            (c) A blow to the back while squatting
    c. Clinical presentation
        (1) Limb appearance (varies with the type of dislocation present)
            (a) With anterior superior dislocations, the limb is slightly abducted, externally rotated, and extended.
            (b) With anterior inferior dislocations, the limb is abducted, externally rotated, and flexed.
        (2) Associated physical findings
            (a) Diminishing femoral or distal pulse (or progressive swelling of the leg) indicates the need for immediate reduction of the hip.
            (b) If the femoral nerve is involved:
                i. Quadriceps function
                ii. Deep tendon reflexes at knee
                iii. Sensation on anteromedial thigh
2. **Posterior: 80%–90% of hip dislocations (Be able to identify on radiograph.)**
    a. Mechanism of injury: most cases result from motor vehicle collisions in which a direct force is applied to the flexed knee (hitting the dashboard) that is transmitted to the femoral head, which is pushed out through the posterior capsule.
    b. Clinical presentation



Posterior Hip Dislocation



Anterior Hip Dislocation

(1) Limb appearance: shortened, adducted, internally rotated, and flexed

(2) Associated physical findings

(a) Evidence of acetabular/femoral fractures

(b) Knee injury

(c) Sciatic nerve injury

i. Muscle function below the knee

ii. Ability to flex the knee

iii. Sensation on posterolateral leg and sole of the foot

3. Treatment

a. Early reduction is required, because avascular necrosis of the femoral head increases in direct proportion to delay in reduction.

b. Immediate closed reduction (using procedural sedation) should be attempted in the emergency department as soon as appropriate radiographs have been obtained to exclude associated injuries. If this is unsuccessful, closed reduction under general anesthesia is indicated. Neurovascular assessment should be performed before and after any reduction attempts.

4. Complications

a. Early complications

(1) Anterior dislocations → femoral artery, vein, and nerve injury

(2) Posterior dislocation → sciatic nerve injury (10% of patients)

b. Late complications (in addition to osteoarthritis)

(1) Anterior dislocation → femoral artery/vein thrombosis → pulmonary embolism and avascular necrosis of the femoral head

(2) Posterior dislocation → avascular necrosis of the femoral head (15%–30% of patients)

D. Hip fractures

1. Classification

a. Intracapsular (femoral head and neck fractures) or

b. Extracapsular (trochanteric, intertrochanteric, and subtrochanteric)

c. This classification system has prognostic value in that intracapsular fractures are far more likely to result in a disrupted vascular supply and subsequent avascular necrosis than extracapsular fractures.

2. **Clinical presentations**
   a. **An elderly patient complains of hip pain after a fall. The patient limps when he or she walks or may be unable to walk. The affected leg is shortened, abducted, and in slight external rotation → femoral neck fracture**
   b. **A patient complains of hip pain after a fall. The leg is shortened and in marked external rotation → intertrochanteric fracture (fracture line between the greater and lesser trochanters)**
   c. **The ability to ambulate does not exclude a fracture; some patients with nondisplaced or minimally displaced fractures may be able to bear weight.**
3. **Diagnostic evaluation**
   a. **Negative plain radiographs do not exclude a fracture.**
   b. **Patients with negative plain radiographs who are unable to ambulate require further evaluation (CT scan, MRI, or bone scan).**
4. **Complications: femoral neck fractures are common in the elderly and are associated with significant complications, the most frequent of which is avascular necrosis (occurs in 15%–35% of patients).**

E. Pediatric hip disorders

1. Classic clinical scenarios
   a. You are examining an infant with some other condition. Because you routinely perform the Ortolani maneuver in all children <1 year old, you detect a "click" on the affected side → congenital hip dislocation. This disorder is much more common in females, particularly first borns.
   b. You are examining a child <4 years old who presents with fever, irritability, and a toxic appearance. The parents tell you that the child limps, and you observe a limp on the affected side when the child walks. With the child lying supine on the cart, you see that the affected leg is flexed, abducted, and externally rotated → septic arthritis of the hip caused by *Staphylococcus aureus*. Ultrasound of the hip reveals an effusion. Arthrocentesis performed by the orthopedic surgeon under fluoroscopic guidance confirms the diagnosis; synovial fluid analysis typically reveals 80,000–200,000 WBCs with a predominance (>75%) of PMNs.
   c. You are examining a nontoxic-appearing child between the ages of 18 months and 12 years who presents with hip, knee, or thigh pain and a limp or inability to bear weight. This child is usually a male and, most typically, 5–6 years old → transient (toxic) synovitis (nonspecific inflammation of the synovium of the hip, often after a nonspecific viral illness). This is a diagnosis of exclusion. Guided aspiration of the hip (performed by an orthopedic consultant) is sometimes needed to distinguish this entity from septic arthritis. Synovial fluid analysis typically reveals 5,000–15,000 WBCs with <25% PMNs.
   d. You are examining a child (frequently a boy) 4–9 years old who presents with a limp. The child may also complain of muscle spasm and groin or hip pain that radiates to the inner aspect of his thigh and knee. The child is afebrile and nontoxic in appearance. Examination of his hip reveals limitation of abduction and internal rotation and spasm. The WBC count and sedimentation rate are normal. Radiograph findings vary with the time course of the disease process. Widening of the joint space and smaller size of the ossific nucleus of the femoral head are the initial findings, followed by development of a subchondral stress fracture line in the femoral head (Caffey crescent line). Subsequent findings include increased density of the femoral head with later fragmentation and distortion → Legg-Calve-Perthes Disease (osteochondrosis of the femoral head). A

technetium bone scan (shows decreased uptake in the femoral head) or MRI (shows low signal intensity in the femoral head) can be used to confirm the diagnosis if doubt remains.

e. You are examining an adolescent or preadolescent (usually a male) who presents with groin pain after activity. He may also complain of hip stiffness, knee pain, and a limp. The build of this youth is classic: he is typically obese with underdeveloped genitalia, or he is tall, thin, and rapidly growing. There is often a history of trivial antecedent trauma. The affected leg is externally rotated and adducted (and may be shortened). Further evaluation reveals tenderness around the hip and decreased range of motion (internal rotation and abduction [full flexion] are limited). Radiographs (AP and bilateral frog-leg lateral views of the hips) confirm the diagnosis → slipped capital femoral epiphysis. Earliest findings (preslippage stage) are irregular widening of the epiphyseal plate, decalcification of the epiphyseal border of the metaphysis, and globular swelling of the joint capsule; with slippage, there is posterior and inferior (medial) displacement of the femoral head and loss of the Shenton line (a curved line formed by the top of the obturator foramen and the inner side of the neck of the femur). Bilateral involvement eventually develops in up to 40% of these children.

2. All of the above-described hip disorders require orthopedic referral.

IV. KNEE INJURIES

A. Essential anatomy

1. There are five stabilizers (two static and three dynamic) of the lateral aspect of the knee.
   a. Static (ligamentous) stabilizers
      (1) Lateral collateral ligament
      (2) Joint capsule
   b. Dynamic (muscular) stabilizers
      (1) Iliotibial tract
      (2) Popliteus muscle
      (3) Biceps femoris muscle
2. There are four stabilizers of the medial aspect of the knee:
   a. Static stabilizers
      (1) Medial collateral ligament (injury to this ligament is much more common than to the lateral collateral ligament)
      (2) Medial joint capsule
   b. Dynamic stabilizers
      (1) Semimembranosus muscle
      (2) Pes anserinus
3. The quadriceps tendon permits extension and is the primary dynamic stabilizer of the knee. It is formed by four muscles:
   a. Rectus femoris
   b. Vastus lateralis
   c. Vastus intermedius
   d. Vastus medialis

4. The cruciate ligaments
   a. Two internal bands extending from the tibia to the femur (one anteriorly, the other posteriorly)
   b. Control anteroposterior and rotatory stability of the knee and prevent hyperextension
   c. Anterior cruciate ligament is the most frequently injured ligament in the knee. It has a rich blood supply, which accounts for the high incidence of hemarthrosis when this ligament is injured.
   d. Posterior cruciate ligament is significantly stronger than the anterior cruciate and collateral ligaments; thus, injury to it is rare and is usually associated with severe knee injuries.

B. Mechanisms of knee injury

1. Valgus (abduction) stress: most common mechanism of injury
   a. Definition: outward stress on lower leg with angulation of the foot away from the midline
   b. Clinical presentation
      (1) Medial collateral ligament (eg, ski injury): 61%
      (2) Anterior cruciate ligament: patient stumbled, caught himself or herself, and now cannot climb steps.
      (3) Medial meniscus: patient complains of knee "locking" in place or clicking (medial meniscus attached to medial collateral ligament).
2. Varus (adduction) stress: uncommon mechanism of injury
   a. Definition: inward stress on lower leg with angulation of the foot toward the midline
   b. Clinical presentation
      (1) Lateral collateral ligament (eg, skier fails to maintain a snow-plow and the tips of his or her skis cross): significant disruption → peroneal nerve injury
      (2) Lateral posterior capsule: knee buckling or "giving way"
      (3) Lateral meniscus: knee "locking" or clicking
3. Hyperextension stress
   a. Definition: backward stress with the knee extended
   b. Usually results in injury to the cruciate ligaments
   c. Clinical presentation
      (1) Anterior cruciate ligament: hyperextension of the knee
      (2) Rupture of the posterior capsule: knee buckling, collapsing, or "giving way"
      (3) Isolated posterior cruciate ligament: blow to the anterior aspect of the tibia with the knee in extension or hyperextension
4. Anterior and posterior forces of the tibia on the femur
   a. Usually occur with the knee in flexion
   b. Clinical presentation
      (1) Anterior cruciate ligament/tibia is forced anteriorly relative to the femur.
      (2) Posterior cruciate ligament/tibia is forced posteriorly relative to the femur.
5. Rotational stress
   a. Definition: rotation of the knee (tibia relative to femur) with the foot firmly planted on the ground
      (1) Internal rotation → ruptures the anterior cruciate ligament

(2) External rotation → disrupts the anterior cruciate and/or the medial capsular and medial collateral ligaments

b. Clinical presentation: a sudden stop and pivot while running; partial ligament ruptures are often more painful than complete tears, and cruciate ligament injuries are more common in women.

C. Hemarthrosis in association with a negative radiograph indicates the presence of one of the following:

1. **Ligamentous tear**
   a. **Hemarthrosis usually develops within 2 hours of injury.**
   b. **Torn anterior cruciate ligament is responsible for 75% of all hemarthroses of the knee.**
2. **Peripheral meniscus tear**
   a. **Effusion develops gradually (6–24 hours after injury).**
   b. **Usually occurs in association with rotational force to a weight-bearing knee**
3. **Osteochondral fracture**
   a. **Hemarthrosis develops immediately.**
   b. **Usually occurs in children, particularly adolescent males**
4. **When aspiration of a hemarthrosis reveals the presence of fat globules (lipohemarthrosis), a fracture is likely.**

D. Physical examination of the knee should include the following tests:

1. Stability tests
   a. Valgus stress in 30° of flexion → instability indicates rupture of the medial collateral ligament.
   b. Valgus stress in extension → instability indicates rupture of the medial collateral ligament as well as potential injury to the cruciate ligaments and posterior capsule.
   c. Varus stress in 30° of flexion → instability indicates rupture of the lateral collateral ligament.
   d. Varus stress in extension → instability indicates rupture of the lateral collateral ligament as well as potential injury to the iliotibial tract, popliteus muscle, and lateral capsule and cruciate ligaments.
   e. Anterior and posterior drawer signs: test the anterior and posterior cruciate ligaments
   f. Lachman test: another test of the anterior cruciate ligament; more sensitive and accurate than the anterior drawer sign
   g. McMurray and Ege tests: aid in diagnosing meniscal tears
      (1) McMurray test → joint line tenderness
      (2) Ege test → pain and/or a click on maximum rotation of the knee with the patient in a squatting position (external rotation → medial meniscus tear; internal rotation → lateral meniscus tear)
2. Patellar compression and apprehension test: pain is elicited with chondromalacia patellae (an overuse syndrome often seen in young women).

E. Knee dislocation (a true orthopedic emergency)

1. **Results from violent trauma, most commonly motor vehicle collisions, vehiclepedestrian collisions, and contact sports**
2. **Knee dislocations are classified according to the direction of the tibial displacement relative to the femur**

a. 5 types: anterior, posterior, medial, lateral, and rotary
b. Anterior and posterior dislocations are the most common (50%–60%).

3. Clinical presentation
   a. Diagnosis is essential, because the incidence of associated injuries is high.
   b. Complete disruption of all major ligaments plus meniscal injury
   c. Popliteal artery injury (21%–32%) is particularly common with anterior and posterior dislocations (up to 40%).
   d. Peroneal nerve injury (25%–35%)
      (1) Paresthesia of the dorsal aspect of the foot
      (2) Diminished dorsiflexion of the foot (foot drop)
      (3) Decreased sensitivity between the first and second toes
      (4) Seen most often with medial and posterolateral dislocations
   e. Tibial nerve injury (plantar foot numbness/paresthesias and weakness with plantar flexion) is less common than peroneal nerve injury.
   f. Proximal tibial fractures
   g. The knee with complete disruption of all major ligaments (and the joint capsule) may exhibit less swelling and pain than the less severely injured knee with a tense hemarthrosis.
   h. The knee may relocate spontaneously, and the diagnosis of dislocation may be missed.
   i. Any patient who presents after trauma with a grossly unstable knee (a tear in three of the four major ligaments of the knee) should be assumed to have a spontaneously reduced dislocation until further evaluation excludes this injury.
   j. Popliteal artery injury may be present even if the pulses are intact or the foot is warm.

4. Treatment
   a. The dislocated knee should be reduced immediately in the neutral position (extension of the knee stretches the popliteal artery) by longitudinal traction in a sedated patient. Do not wait for radiographic confirmation.
   b. The popliteal, dorsalis pedis, and tibialis posterior pulses should be checked before and after reduction.
   c. After reduction, the knee should be immobilized in a posterior splint in 15° flexion.
   d. A Doppler flow ultrasound, CT angiography, or standard angiography should be strongly considered in all cases in consultation with a vascular surgeon, even if pulses are present.
      (1) Distal pulses may still be present but a vascular injury (eg, intimal flap injury, pseudoanuerysm or partial intimal tear) may be present and lead to vascular complications if not found early.
      (2) If immediate surgical intervention is indicated, arteriography should be performed in the operating room, because performance in the radiology department is associated with an additional delay of 1–2 hours before surgery.
   e. The peroneal and tibial nerves should be tested before and after reduction.
   f. Immediate orthopedic (and possibly vascular) surgery consult is indicated whenever this injury is suspected.
   g. Immediate surgical intervention is indicated for:
      (1) Popliteal artery injuries (arterial repair should be accomplished within 6 hours to avoid the complications of prolonged ischemia).
      (2) Open dislocations
      (3) Irreducible dislocations

F. Rupture of the quadriceps mechanism

1. May result from one of the following injuries:
   a. Quadriceps tendon rupture
   b. Patellar tendon rupture
   c. Patellar fracture
   d. Avulsion of the tibial tuberosity
2. Clinical presentation
   a. With a complete disruption, the patient can stand but cannot walk or extend the knee; on physical examination, there is diffuse swelling around the knee and a palpable deficit.
   b. With incomplete disruption, active extension is weak and painful.
3. Treatment
   a. Early orthopedic consult
   b. Complete ruptures → early surgical repair
   c. Partial tear → immobilization in full extension

G. Patellar subluxation/dislocation

1. Mechanisms of injury
   a. Sudden flexion and external rotation of the tibia on the femur with concomitant contraction of the quadriceps
   b. Direct blow to the patella with the knee in flexion or extension
2. Lateral subluxation/dislocation is the most common.
3. Clinical presentation: adolescent females with chronic patellofemoral anatomic abnormalities such as genu valgum (knock-knees), patellae alta (high-riding patellae), a large Q (quadriceps) angle, or generalized joint laxity
4. Diagnostic evaluation: obtain AP and lateral radiographs of the knee before and after reduction to exclude associated osteochondral fracture of the patellae or lateral femoral condyle.
5. Treatment
   a. Patellar subluxations often reduce spontaneously.
   b. Reduction is accomplished by flexing the patient's hip and applying gentle, medially directed pressure over the lateral aspect of the patellae while extending the knee.
   c. Immobilize in full extension
   d. Crutches: patient should be non-weight-bearing.
   e. Orthopedic referral for possible surgical intervention
   f. Isometric quadriceps exercise should be started as soon as the patient can do them without pain.

H. Patellar fracture

1. One of several possible injuries to the extensor mechanism of the knee
2. Mechanisms of injury
   a. Direct blow (most common), eg, against the dashboard of a car
   b. Forceful contraction of the quadriceps muscles
   3. Transverse fractures are the most common (50%–80% of cases).
   4. Clinical presentation: patients typically present with tenderness and swelling over the patella and limited, painful knee extension (if the extensor mechanism is intact) or inability to extend the knee (if the extensor mechanism is torn).

5. Diagnostic evaluation: radiograph evaluation should include AP, lateral, and sunrise (skyline) views.
6. Treatment
   a. Nondisplaced or minimally displaced and the extensor mechanism is intact
      (1) Immobilize in full extension, partial weight-bearing advancing to full weight-bearing as tolerated
      (2) Orthopedic referral
   b. If the fracture fragments are widely displaced (>3 mm) or there is loss of extensor function
      (1) Orthopedic referral for surgical intervention is required.
      (2) When fracture fragments are widely separated, associated concomitant knee joint injury is frequently present.

I. Tibial plateau fracture (most common fracture of the knee)

1. Most common mechanism of injury is a strong valgus stress with axial loading.
2. Most condylar fractures (55%–70%) involve the lateral plateau.
3. Vascular complications are common.
   a. Fracture fragments in the subcondylar area → popliteal artery
   b. Displaced fracture of the lateral condyle → anterior tibial artery

J. Baker cyst

1. An inflammation of the semimembranosus or medial gastrocnemius bursa produced by protrusion of the synovial membrane through the posterior aspect of the knee capsule
2. In adults, these cysts are usually due to an intra-articular abnormality of the knee, such as chronic inflammatory arthritis or a torn meniscus. In children, however, these cysts appear to be a primary lesion.
3. Clinical presentation
   a. History of intermittent swelling behind the knee and may complain of local discomfort
   b. Examination reveals a tense and occasionally painful fluid-filled sac in the medial aspect of the popliteal fossa.
   c. Rupture of the cyst with inferior dissection of the synovial fluid causes calf pain and swelling, often simulating thrombophlebitis or a deep vein thrombosis of the calf.
4. Treatment
   a. In adults, management is directed toward correction of the underlying joint pathology.
   b. In children, treatment is generally unnecessary, because most of these cysts resolve on their own in 1–2 years.

K. Osgood-Schlatter disease (traumatic tibial apophysitis)

1. Due to repetitive microscopic injury that produces inflammation of the apophysis of the tibial tubercle and leads to partial avulsion and separation of the tibial tubercle
2. Clinical presentation
   a. Commonly seen in adolescents (particularly males) who are active in sports
   b. Pain and swelling in the region of the tibial tuberosity that is increased with forceful leg extension (climbing stairs, running, jumping); often bilateral (25% of cases)
3. Treatment: ice, NSAIDs, and decreased participation in sports

L. Chondromalacia patellae (patellofemoral pain syndrome/patellar malalignment syndrome)

1. Most common cause of knee pain
2. Usually occurs in young adults (particularly females)
3. Often bilateral
4. Causes include patellofemoral malalignment, recurrent patellar subluxation/dislocation, and excessive knee strain (athletes)
5. Clinical presentation
   a. Intermittent peripatellar pain that is exacerbated by prolonged sitting and climbing steps
   b. Pain with compression of the patellae into the femoral groove (patellar compression test) and with knee extended and quadriceps tightened
   c. Quadricep muscle contraction in anticipation of pain when the patella is displaced laterally while the knee is extended and relaxed (patellar apprehension test)
   d. Findings of patellar malalignment (Q angle >20°) may also be present. The Q angle is formed by the intersection of two lines: one drawn from the anterior superior iliac spine to the center of the patellae and the second drawn from the center of the patellae through the tibial tuberosity (the normal Q angle is 15°).
6. Treatment: rest and NSAIDs, followed by isometric quadricep-strengthening exercises (quadricep contraction with adduction)

V. LOWER LEG INJURIES

A. The soft tissue surrounding the tibia and fibula is divided into four compartments.

1. Anterior compartment
   a. Dorsiflexor muscles of the ankle and foot (tibialis anterior, extensor digitorum longus, extensor hallucis longus, and peroneus tertius)
   b. Anterior tibial artery
      (1) Most frequently injured structure in this compartment
      (2) Injury occurs secondary to fractures of the:
         (a) Lateral tibial plateau
         (b) Proximal third of the tibia
   c. Deep peroneal nerve
      (1) Provides sensation to the first dorsal web space of the foot
      (2) May be injured because of fracture of the lateral tibial plateau
2. Lateral (peroneal) compartment
   a. Everter and plantar flexor muscles of the foot (peroneus brevis and longus muscles)
   b. Superficial peroneal nerve
      (1) Provides sensation to the dorsum of the foot
      (2) Frequently injured because of fracture of the fibular neck or proximal shaft
3. Deep posterior compartment
   a. Tibialis posterior, flexor digitorum longus, and flexor hallucis longus muscles
   b. Posterior tibial and peroneal arteries

c. Posterior tibial nerve provides sensation to the sole of the foot.

4. Superficial posterior compartment
   a. Gastrocnemius, plantaris, and soleus muscles
   b. Sural nerve provides sensation to the lateral aspect of the foot and the distal calf.

B. Anterior compartment syndrome

1. **More common than the lateral or posterior compartment syndromes**
2. **Usually occurs in association with proximal tibial fractures but can result from prolonged strenuous exercise (particularly in runners) or other causes**
3. **Clinical presentation**
   a. **Anterior tibial pain that is:**
      (1) **Out of proportion to the injury (one of the earliest findings)**
      (2) **Increased with active dorsiflexion of the foot (one of the earliest findings)**
      (3) **Increased with passive plantar flexion of the foot**
   b. **Decreased two-point sensory discrimination (most consistent finding)**
   c. **Paresthesia or hypesthesia in the distribution of the deep peroneal nerve (early finding)**
   d. **Weak dorsiflexion of the ankle and toes**
   e. **Palpable tenseness and tenderness of the compartment**
   f. **Pallor of the skin (late finding)**
   g. **Pulselessness or reduced distal pulses (a very late and frequently absent finding). The presence of peripheral pulses does not exclude increased intracompartmental pressure; pulses often remain intact.**
4. **Treatment**
   a. **Remove constrictive dressing or cast (if present).**
   b. **Obtain immediate orthopedic consult and measure intracompartmental pressure.**
   c. **Surgical decompression via fasciotomy is indicated if the intracompartmental pressure is >30 mmHg.**

C. Stress fractures of the fibula

1. Result from overtraining and usually occur at the distal fibula
2. Patients present with pain exacerbated with ambulation.
3. Initial radiographs are usually negative.
4. Treatment is symptomatic.

VI. ANKLE INJURIES

A. Essential anatomy

1. Three groups of ligaments unify the bony structures of the ankle
   a. Medial collateral (deltoid) ligament
      (1) Provides medial support to the ankle
      (2) Originates from the medial malleolus and inserts on the navicular and talus
   b. Anterior talofibular, calcaneofibular, and posterior talofibular ligaments → provide lateral support.

c. Ligaments of syndesmosis → bind the lower portions of the tibia and fibula together.
   (1) Anterior and posterior tibiofibular ligaments
   (2) Transverse tibiofibular ligament
   (3) Interosseous ligament (provides the strongest bond between the tibia and fibula at the ankle joint)

2. Four groups of muscles traverse the ankle joint
   a. Anteriorly → dorsiflexion of the ankle
      (1) Tibialis anterior
      (2) Extensor digitorum longus
      (3) Extensor hallucis longus
   b. Medially → inversion of the foot
      (1) Tibialis posterior
      (2) Flexor digitorum longus
      (3) Flexor hallucis longus
   c. Posteriorly → plantar flexion
      (1) Gastrocnemius
      (2) Plantaris
      (3) Soleus
   d. Laterally → eversion and plantar flexion
      (1) Peroneus longus
      (2) Peroneus brevis

B. Sprains (ligamentous injury)

1. Lateral collateral ligament sprains
   a. Most common (85%–90%)
   b. Result from inversion and internal rotation of the plantarflexed foot
   c. Ligamentous injury occurs in sequence, starting anteriorly with the anterior talofibular ligament and progressing posteriorly to the calcaneofibular ligament, followed by the posterior talofibular ligament.
   d. The most frequently injured ligament → anterior talofibular
2. Medial collateral ligament sprains are uncommon.
   a. Result from eversion and external rotation of the foot
   b. Often associated with either a fracture of the fibula or a tear of the tibiofibular syndesmosis
3. Distal tibiofibular syndesmotic ligament sprains (high ankle sprains) are also uncommon.
   a. Result from dorsiflexion and eversion forces with an axial load
   b. Have a prolonged recovery period
4. Classification is based on the clinical presentation and the degree of instability present (as demonstrated by stress testing).
   a. First degree
      (1) Mild localized tenderness
      (2) Minimal swelling
      (3) No instability or functional loss

(4) Able to bear weight
(5) No abnormal motion

b. Second degree
(1) Moderate swelling and tenderness
(2) Moderate functional loss
(3) Increased pain with stress testing

c. Third degree
(1) Marked tenderness
(2) Egg-shaped swelling over the affected ligament(s) within 2 hours of injury
(3) Significant functional loss
(4) Inability to bear weight
(5) Resistance to motion of the foot
(6) Positive stress test

5. Stress tests for ankle stability are useful in confirming specific ligamentous injury and in distinguishing second from third-degree sprains.
a. Anterior drawer test: abnormal movement indicates a rupture of at least the anterior talofibular ligament.
b. Inversion stress (or talar tilt) test
(1) Needs to be done only if the anterior drawer test is positive
(2) Abnormal movement indicates rupture of both the anterior talofibular and the calcaneofibular ligaments.
c. External rotation test
(1) Pain at the syndesmosis or a sensation of lateral talar movement indicates injury to the distal tibiofibular syndesmotic ligament.
(2) Accurate results require the presence of good muscle relaxation obtained with application of ice or infiltration of a local anesthetic.

6. Associated injuries
a. Maisonneuve fracture
(1) A proximal fibular fracture that occurs in association with rupture of the deltoid ligament or an avulsion fracture of the medial malleolus at the insertion site of the distal talofibular ligament
(2) This diagnosis should be suspected in patients presenting with a history of eversion injury and significant medial malleolar tenderness and swelling.
b. Osteochondral fracture of the talar dome
c. Avulsion fracture of the fifth metatarsal occurs with flexioninversion injuries of the foot.
d. Peroneal tendon dislocation or subluxation

7. Treatment
a. Determined by the degree of injury; all sprains should initially be managed with ice, elevation, and compression. The management of third-degree sprains, however, is controversial (operative versus nonoperative) and requires orthopedic consult.
b. First- and most second-degree sprains
(1) Ice, elevation, immobilization in a protective device (air cast, brace, splint, Unna boot), and crutches as needed.

(2) As pain and swelling decrease, this should be followed by graded exercises to increase dorsiflexion and peroneal strength, weight bearing as tolerated, and reevaluation by the primary care provider.

c. Severe second-degree sprains
   (1) Ice, elevation, splint immobilization (sugar-tong or posterior mold), and crutches
   (2) Early orthopedic reevaluation for possible casting

d. Third-degree sprains
   (1) Ice, elevation, splint immobilization, and crutches
   (2) Orthopedic evaluation within 24 hours (or while in the emergency department)
   (3) Some of these patients, depending on their level of athletic involvement and other associated injuries, may be considered for primary surgical repair.

C. Ankle dislocations

1. Described according to the direction of talar displacement relative to the tibia; five types:
   a. Anterior
   b. Posterior (common)
   c. Medial (common)
   d. Lateral
   e. Superior (diastasis) (uncommon)
2. Pure ankle dislocations are uncommon; most occur in association with malleolar fractures.
3. Treatment
   a. All of these dislocations require immediate neurovascular assessment and emergent orthopedic consult in the emergency department.
   b. If there is any evidence of neurovascular compromise or skin tenting, reduction should be accomplished immediately (before radiographic evaluation). If, however, these findings are absent, radiographs should be obtained before reduction to exclude the presence of associated fractures.
   c. After administration of adequate analgesia or conscious sedation, promptly reduce the dislocation with gentle in-line traction.
   d. Once reduction has been accomplished, reassess the patient's neurovascular status, order radiographs after reduction, and place the patient in a posterior splint.

D. Tendon injuries

1. Achilles tendon rupture
   a. Common in sedentary, middle-aged men (mean age 35 years) engaging in weekend athletic activities
   b. Up to 25% of cases are initially misdiagnosed as ankle sprains.
   c. Mechanism of injury
      (1) Forceful dorsiflexion of the foot with the ankle in a relaxed state
      (2) Direct trauma to a taut tendon
      (3) An extra stretch applied to a taut tendon
   d. Clinical presentation
      (1) The patient gives a history of sudden excruciating pain at the back of the ankle that lessens instantly, and often reports having heard or felt a "pop" or "snap."
      (2) Physical examination

(a) Swelling of the distal calf
(b) A palpable defect in the tendon 2–6 cm proximal to its calcaneal insertion site
(c) Weak plantar flexion when compared with the uninjured leg

e. Diagnostic evaluation
(1) Diagnosis is made with the Thompson test (also called Simmonds test), which is performed with the patient in the prone position.
(a) Normal function: squeezing the calf produces plantar flexion of the foot.
(b) With a complete tear, plantar flexion will not occur.
(c) Ultrasound or MRI can be used to confirm the diagnosis if doubt remains, but this is usually unnecessary.

f. Treatment
(1) Initial management
(a) Ice, elevation, analgesia, immobilization in a posterior splint in passive equinus (plantar flexion), and crutches
(b) Orthopedic consult within 48–72 hours
(2) Definitive care (casting versus surgical repair) remains controversial; it is usually determined by the patient's age, activity level, and underlying medical conditions. Surgical repair appears to be associated with a better outcome (increased strength and mobility, and decreased incidence of re-rupture) but is typically reserved for young athletic patients.

2. Peroneal tendon subluxation/dislocation
a. Results from a tear of the superior peroneal retinaculum from its attachment on the fibula. This allows the peroneus brevis and longus tendons to sublux anteriorly over the lateral malleolus.
b. Often misdiagnosed as an ankle sprain
c. Mechanism of injury: forced dorsiflexion of the ankle with reflex contraction of the peroneal muscles
d. Clinical presentation
(1) The patient typically complains of pain and a clicking or slipping sensation at the back of the ankle.
(2) Diagnosis is made by physical examination.
(a) Tenderness, swelling, and ecchymosis at the posterior aspect of the lateral malleolus
(b) Tensing of the peroneal muscles intensifies the pain.
(c) Eversion is weak.
(d) Anterior subluxation of the tendons with the patient's foot dorsiflexed and everted against resistance confirms the diagnosis.
e. Plain radiographs may reveal a small avulsion fracture of the lateral ridge of the distal fibula (pathognomonic for this injury) in 50% of cases.
f. Treatment: splint in mid plantar flexion, and refer to orthopedist for possible surgical repair.

E. Ankle injuries in children

1. Because ligaments are stronger than bone in children, fractures at the epiphyseal plate are more common than ligamentous injuries.

2. These fractures usually result from indirect forces and are classified according to the Salter-Harris classification system (see pages 441–442).
3. Tillaux fracture
   a. A Salter III fracture of the lateral distal tibia commonly seen in adolescents participating in sporting events
   b. Consider this diagnosis if the history is suggestive and there is swelling over the anterior ankle.

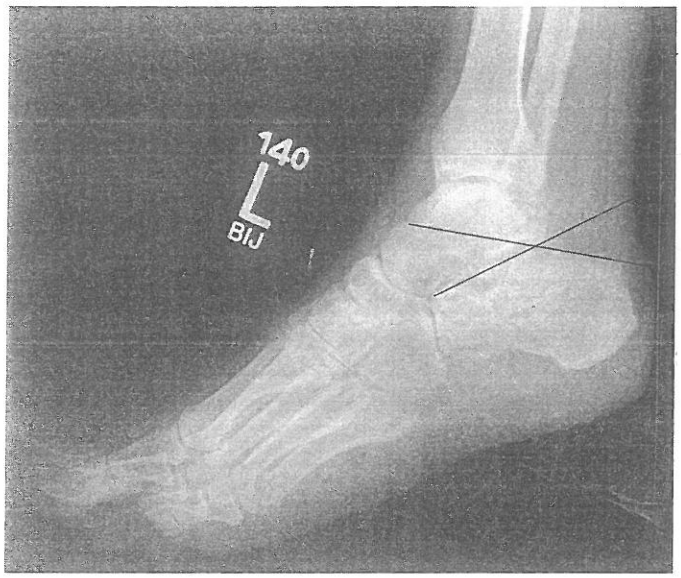
VII. FOOT INJURIES

A. Essential anatomy

1. The foot is divided into three parts:
   a. Hindpart (calcaneus and talus)
   b. Midpart (navicular, cuboid, cuneiforms)
   c. Forepart (metatarsals and phalanges)
2. The hindpart is separated from the midpart by Chopart joint; the mid part is separated from the forepart by Lisfranc joint.
3. First metatarsal head bears twice the weight of the other metatarsals.
4. During the push-off phase, the second and third metatarsals bear the most weight; hence, they are prone to stress fractures.

B. Fractures

1. Calcaneus
   a. The calcaneus is the most frequently fractured tarsal bone. (The talus is the second most commonly fractured.)
   b. A calcaneal fracture is usually due to a compression injury (eg, fall from a height with the patient landing on his or her feet).
   c. Clinical presentation
      (1) Examination reveals swelling, tenderness, and ecchymosis of the hindfoot and inability to bear weight on the fracture.
      (2) 10% are bilateral, and another 10% are associated with compression fractures of the dorsolumbar spine.
      (3) 26% are associated with other injuries to the lower extremities.
      (4) Whenever a calcaneal body fracture is diagnosed, Bohler angle should be measured to determine whether or not the fracture is depressed. This is done by measuring the intersection of two lines on the lateral radiograph. One is drawn from the superior margin of the posterior tuberosity of the calcaneus and extended through the superior tip of the posterior facet. The other line is drawn from the superior tip of the anterior process and extended through the superior tip of the posterior facet. This angle normally measures 20°–40°. If the angle is <20°, the fracture is depressed.



Courtesy of Michael C. Bond, MD

d. Treatment

(1) Intra-articular or displaced calcaneal fractures: management remains controversial (nonoperative versus immediate surgical reduction); obtain emergent orthopedic consult in the emergency department.

(2) Nondisplaced or minor extra-articular fractures: ice, elevation, immobilization in a posterior splint, crutches; obtain orthopedic consult and arrange for early follow-up on an outpatient basis.

2. Lisfranc (tarsometatarsal) fracture/dislocation

a. Mechanisms of injury

(1) Axial load: a fall on the plantar-flexed foot

(2) Compressive forces: crush injury

(3) Rotational forces: twisting of the body around a fixed foot

b. Clinical presentation

(1) Examination reveals significant midfoot swelling and pain, decreased range of motion, and inability to bear weight.

(2) Paresthesias of the midfoot may also be present.

c. Diagnostic evaluation: radiographs

(1) The key to making this diagnosis is to look for the normal alignment along the medial aspect of the middle cuneiform with the medial aspect of the base of the second metatarsal; any disruption of this alignment is indicative of a dislocation.

(2) The second metatarsal functions as the primary stabilizing force of the joint; thus, a fracture through the base of the second metatarsal (Fleck sign) is indicative of a disrupted Lisfranc joint.

(3) Separation between the base of the first and second metatarsals is highly suggestive of subluxation and is the most reliable finding.

d. Treatment

(1) Involves either closed reduction under general anesthesia, or open reduction and surgical fixation

(2) Early orthopedic consult in the emergency department is indicated.

3. Jones fracture (diaphyseal fracture of the fifth metatarsal)
   a. A transverse fracture of the proximal diaphysis of the fifth metatarsal
   b. In children, the proximal physis of the fifth metatarsal may be mistaken for a fracture.
   c. Mechanism of injury: a forceful load applied to the ball of the foot laterally (such as a pivot)
   d. Unlike avulsion fractures of the base of the fifth metatarsal (Dancer fracture), these fractures are slow to heal and are associated with a high incidence of delayed union or nonunion.
   e. Treatment
      (1) Nondisplaced fractures are usually managed with immobilization in a non-weight-bearing short-leg cast.
      (2) Displaced fractures (and fractures in athletes) are typically managed surgically.
      (3) Orthopedic follow-up is required.

ORTHOPEDIC EMERGENCIES: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: An elderly man with a history of prosthetic hip replacement presents complaining of inability to walk or move the hip. The pain started with minimal trauma.

Physical examination: On physical examination, the leg is shortened, abducted, and internally rotated.

What is the diagnosis?

Scenario B

Presentation: A middle-aged "weekend warrior" felt a snap or pop in his knee after a jump in a weekend game of basketball.

Physical examination: On physical examination, the patient can stand but is unable to walk or extend the knee. The patella is high riding.

What is the diagnosis?

Scenario C

Presentation: The patient presents after a fall onto the foot or a twisting injury. Pain on the medial side of the ankle increases with weight bearing.

Physical examination: A careful examination of the proximal fibula elicits pain with compression of the fibula or palpation of the fibula head.

What is the diagnosis?

Scenario D

Presentation: A young football player is brought in from the field complaining of knee pain after taking a hit during the game. Bystanders and the patient report that the knee appeared deformed.

Physical examination: An effusion is noted in the knee, along with multidirectional laxity (laxity with valgus, varus, anterior and posterior stress of the ligaments).

What is the diagnosis?

Scenario E

Presentation: Parents bring in a 3-year-old child, reporting arm pain. The child refuses to use the affected arm.

Physical examination: The elbow is tender over the radial head, but there is no bruising, swelling, or deformity.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: posterior prosthetic hip dislocation

Diagnostic evaluation: Pelvis and hip radiographs show the hip posteriorly dislocated. Care should be taken to ensure that there is no acetabular or pelvic fracture.

Scenario B

Diagnosis: quadriceps rupture

Diagnostic evaluation: Radiographs of the knee may show a high-riding patella (can also be normal). A joint effusion can often be seen.

Scenario C

Diagnosis: Maisonneuve fracture

Diagnostic evaluation: Ankle radiographs show a fracture of the medial malleolus, possibly with increased space between the malleolus and talar dome. Tibia/fibula radiographs demonstrate a proximal fibula fracture.

Scenario D

Diagnosis: knee dislocation

Diagnostic evaluation: It is not unusual for the leg to look completely normal except for an effusion. Evaluation should consist of serial examinations of the leg with measurement of an ankle-brachial index (ABI). Normally, ABIs do not definitively exclude arterial injury, because an intimal tear can initially be normal until a thrombosis forms and occludes the artery. Knee radiographs (AP and lateral) should be obtained to exclude fractures. These patients are at risk of compartment syndrome and injury to the popliteal artery. Consider obtaining Doppler ultrasound or a CT arteriogram (or angiogram) of the popliteal artery.

Scenario E

Diagnosis: nursemaid elbow (radial head subluxation)

Diagnostic evaluation: There may be a history of the arm being pulled or yanked. Radiographs are generally not required; however, PA and lateral views of the elbow should be obtained if there is a concern for fracture or dislocation or if there is swelling, bruising, or crepitus on examination.

Management: Treatment consists of reduction of the dislocated radial head by supinating and if necessary flexing the elbow while applying some gentle pressure to the radial head. If supination is not effective, reduction may be attempted by hyperpronating the forearm and flexing the elbow.

MUSCULOSKELETAL DISORDERS (NONTRAUMATIC)

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MUSCULOSKELETAL DISORDERS (NONTRAUMATIC): SELF-ASSESSMENT QUESTIONS

1. Empiric antibiotic coverage from human bite cellulitis associated with involvement of the metacarpophalangeal joint (clenched fist injuries) should be selected to cover all the following pathogens except:

   (a) Anaerobic streptococci
   (b) *Staphylococcus aureus*
   (c) *Eikenella corrodens*
   (d) *Pasteurella multocida*

2. Synovial fluid analysis with a WBC count of 3,500/mm^3 and PMNs of 60% is most likely related to which disease process?

   (a) Septic arthritis
   (b) Osteoarthritis
   (c) Pseudogout
   (d) Traumatic arthritis

3. "Red flags" in a patient presenting with back pain that require further evaluation in the emergency department include all the following *except:*

   (a) History of breast cancer
   (b) Fever
   (c) Acute onset in a patient aged 30
   (d) Bladder retention

4. A patient presents with finger pain and has a red, swollen index finger with vesicles present and itching for 3 days. This presentation is consistent with a diagnosis of:

   (a) Herpetic whitlow
   (b) Felon
   (c) Paronychia
   (d) Spider bite

5. All of the following are Kanavel cardinal signs of flexor tenosynovitis except:

   (a) Pain with passive extension of the finger
   (b) Lack of capillary refill
   (c) Finger held in slight flexion
   (d) Fusiform swelling of the digit ("sausage digit")

6. All of the following can lead to Volkmann ischemic contracture of the forearm except:

   (a) Arterial injection of drugs
   (b) Burns
   (c) Boxer's fracture
   (d) Extravasation of blood into the forearm

7. Rhabdomyolysis may occur when _____.

   (a) ATP stores become depleted.
   (b) There is damage to the sarcolemma of the cell.
   (c) The calcium channels, pumps, and exchangers fail to function.
   (d) All of the above
   (e) None of the above

8. Treatment for rhabdomyolysis should be directed at:

   (a) Aggressive fluid repletion
   (b) Urine alkalinzation
   (c) Correcting electrolyte derangements
   (d) All of the above

9. The most common pathogen responsible for epidural abscess is _____.

   (a) *Pseudomonas* spp
   (b) *Escherichia coli*
   (c) *Staphylococcus aureus*
   (d) *Mycobacterium tuberculosis*

10. A patient presents with acute onset of back pain and complains of pain extending down the leg from the thigh to the medial aspect of the foot. A decreased knee jerk is present on examination. This correlates to nerve root involvement at which level of the spine?

    (a) L2
    (b) L3
    (c) L4
    (d) L5

11. Which of the following statements about rheumatoid arthritis is true?

    (a) It causes symmetric monoarticular joint pain.
    (b) It affects all joints of the hands, wrist and elbow in an additive manner.
    (c) It is a migratory polyarthritis.
    (d) It has a genetic predisposition to the HLA-DR4 haplotype.

ANSWERS

1. d	4. a	7. d	10. c
2. c	5. b	8. d	11. d
3. c	6. c	9. c	

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. ARTHRITIS

A. Classification

1. Arthritis can be produced by many conditions and may be classified in several ways.
2. Classification of arthritides by pattern of joint involvement (polyarticular, monarticular, migratory) is useful, but there is some overlap.

B. Conditions associated with symmetric polyarticular joint pain

1. **Rheumatoid arthritis**
   a. **Most commonly affects women; there is a genetic predisposition related to HLA-DR 4 haplotype.**
   b. **Most commonly affects the hand (metacarpophalangeal and proximal interphalangeal joints, spares distal interphalangeal joints), wrist, and elbow in an additive manner**
   c. **Extra-articular complications may occur, including tenosynovitis, pericarditis, subcutaneous nodules, vasculitis of the skin, pulmonary fibrosis, mononeuritis multiplex, Sjögren syndrome, and Felty syndrome. Long-standing rheumatoid arthritis may lead to degeneration of the transverse ligament of the C1–C2 junction, which requires precautions during endotracheal intubation.**
   d. **Treatment is with salicylates/NSAIDs, brief courses of corticosteroids or long-term with antimalarial agents, gold, and methotrexate.**
2. Systemic lupus erythematosus
   a. Women 15–40 years of age are most commonly affected.
   b. May be genetic (associated with HLA genotypes), environmental (exposure to UV light), or drug-induced
   c. Arthralgias and arthritis are the most common presenting complaints; both small and large joints are affected.
   d. An erythematous malar ("butterfly") rash is a typical feature and often accompanies joint involvement.
3. Rheumatic fever
   a. A migratory polyarthritis
   b. Primarily affects the large joints of the extremities (knees, ankles, elbows, and wrists)
4. Bacterial endocarditis (migratory)
5. Hepatitis B (migratory)
6. Rubella
   a. Occurs with both natural and vaccine-induced rubella
   b. Most commonly affects young women
   c. Finger, wrist, and knee involvement is typical.

C. Conditions associated with asymmetric polyarticular joint pain

1. Reiter syndrome (reactive arthritis)
   a. Usually affects young males 15–35 years old; the HLA-B27 antigen is found in up to 75% of these patients.
   b. Clinical presentation
      (1) Classic triad: urethritis, conjunctivitis, and arthritis
      (2) This syndrome has also been documented to occur after dysentery and cervicitis.

(3) Oligoarthritis usually develops 1–6 weeks after an episode of urethritis (due to *Chlamydia*) or dysentery (due to *Salmonella, Shigella, Yersinia,* or *Campylobacter*).

(4) The weight-bearing joints of the lower extremities are most commonly affected (usually four or five).

c. Must be differentiated from gonococcal arthritis

2. Gonococcal arthritis

a. A migratory arthritis that primarily affects young adults, particularly women

b. Clinical presentation

(1) The wrists, fingers, knees, and ankles are most affected.

(2) An associated rash (erythematous macules or pustules with a necrotic or purpuric center), which is distributed primarily on the extremities, accompanies the arthritis in two-thirds of patients (arthritis/dermatitis syndrome).

c. Cervical, urethral, rectal, and pharyngeal cultures should be obtained.

d. Initial treatment is with parental third-generation cephalosporins.

3. Henoch-Schonlein purpura

a. Most commonly occurs in children 4–11 years old

b. Clinical presentation

(1) Classic triad: migratory arthritis, palpable purpuric rash on the lower extremities/buttocks, and colicky abdominal pain

(2) Ankles and knees are most commonly affected.

4. Lyme disease

a. An oligoarthritis that typically affects the large joints (particularly the knees); it can be migratory and usually occurs in those patients not treated with appropriate antibiotics.

b. Arthritic symptoms develop in Stage III of the disease (months to years after the initial infection).

D. Conditions associated with monoarticular joint pain

1. Septic arthritis (a medical emergency)

a. *Staphylococcus aureus* is the most common cause. There is increasing incidence of methicillin-resistant *S aureus* (MRSA).

b. In most adults, the knee is the most commonly affected joint. In IV drug abusers, however, the most common sites are the sacroiliac, sternoclavicular, and intervertebral joints. In children, the knee and hip are most commonly affected.

c. High-risk populations: IV drug users, immunocompromised patients, diabetics, alcoholics, patients with chronic arthritis or skin infections, or after intra-articular injections or prosthetic implants.

d. Treatment aimed at likely causative agents and adjusted according to Gram stain and culture results. Initial treatment in adults is a first-generation cephalosporin plus vancomycin for suspected gram-positive organisms and a third-generation cephalosporin for gram-negative bacilli.

2. Gout

a. A disease of middle-aged men and elderly adults

b. Caused by deposition of uric acid crystals

c. Usually affects the lower extremities, particularly the great toe ("podagra")

d. **Evaluation of synovial fluid reveals needle-shaped crystals that exhibit negative birefringence under polarized light.**

e. **Acute treatment initially includes NSAIDs and colchicine orally, or prednisone for resistant cases. Corticotropin IM in a single dose may also be used, particularly in elderly patients.**

3. **Pseudogout**

a. **Primarily affects men and women ≥60 years old**

b. **Caused by deposit of calcium pyrophosphate crystals**

c. **Most commonly affected joint is the knee, followed by the wrist, ankle, and elbow.**

d. **Treatment mirrors that of gout.**

e. **Diagnostic evaluation**

(1) **Evaluation of synovial fluid reveals rhomboid-shaped crystals that exhibit weakly positive birefringence under polarized light (appears yellow when light is perpendicular to the crystal).**

(2) **Radiographs of the affected joint may reveal calcification of the cartilage (chondrocalcinosis).**

4. Osteoarthritis (degenerative joint disease)
5. Trauma
6. Aseptic arthritis
7. Hemarthrosis
8. Patients with monarticular joint pain should always be considered to have an infectious arthritis until proved otherwise.

E. Conditions associated with migratory arthritis

1. Rheumatic fever
2. Bacterial endocarditis
3. Hepatitis B
4. Septicemia (gonococcal, meningococcal, streptococci, staphylococci)
5. Henoch-Schonlein pupura
6. Lyme disease
7. Serum sickness
8. Pulmonary infections (mycoplasmosis, histoplasmosis, coccidiomycosis)

F. Synovial fluid analysis

1. **The most important diagnostic study in evaluation of an acute arthritis**
2. **Mnemonic for laboratory analysis: "CAPS"**

Culture, cell count, crystals (If there is only enough fluid for one test, it should be culture and sensitivity.)

Appearance

Protein

Sugar, stain (Gram stain)

3. **Crystals**

a. **Gout: negatively birefringent (urate) and needle-shaped**

b. **Pseudogout: positively birefringent (calcium pyrophosphate) and rhomboid-shaped**

4. **WBC count**
   a. **<200/mm³ with <25% PMNs is normal.**
   b. **200–2,000/mm³ with <25% PMNs is noninflammatory (osteoarthritis, traumatic arthritis).**
   c. **2,000–50,000/mm³ with 50%–75% PMNs is inflammatory (rheumatoid arthritis, gout, pseudogout).**
   d. **5,000 to >50,000/mm³ (usually >40,000) with >75% PMNs is septic.**
5. **Glucose is significantly decreased (relative to serum glucose) in both septic and rheumatoid arthritis.**
6. **Gram stain is usually positive, except for in gonococcal infection, in which it is positive in only 25% of cases.**

II. TENDINITIS AND TENOSYNOVITIS

A. General facts

1. Inflammation of flexor or extensor tendons of the hand from repetitive motion
2. Indicated by tenderness on palpation or pain with active or passive range of motion
3. Management is splinting in position of function, elevation, and NSAIDs.

B. Stenosing tenosynovitis or "trigger finger"

1. Scarring or inflammation for prolonged periods of time causes nodules to form on the flexor tendon sheaths.
2. Causes friction and "catching" of the finger at the A1 pulley at the volar crease at the base of each digit
3. Finger "snaps" into extension but may be locked in flexion.
4. Treatment by depot steroid injection or surgical division of the A1 pulley

C. De Quervain stenosing tenosynovitis

1. Due to excessive use of the thumb, the extensor pollicis brevis and abductor pollicis tendons become inflamed.
2. Causes pain along radial aspect of the wrist that extends to the forearm
3. Diagnosis is confirmed by a positive Finkelstein test (thumb is grasped in a closed fist and the wrist is ulnarly deviated, producing a sharp pain along the tendon).
4. Management is immobilization of the thumb and wrist, range of motion exercises, and NSAIDs.

D. Carpal tunnel syndrome

1. Peripheral neuropathy involving the median nerve in the carpal tunnel or canal
2. Swelling of tendons in the canal causes compression of the median nerve, which triggers paresthesias in the palmar aspect of the thumb, the index and long fingers, and the radial aspect of the ring finger.
3. Symptoms include night pain, burning, and tingling in the above-mentioned distribution, and numbness when the wrist is in prolonged flexion.
4. Typically is triggered by overuse syndromes but may occur with direct trauma, diabetes, rheumatoid arthritis, and edematous states such as pregnancy or heart failure.

5. Tinel sign (paresthesias triggered by tapping over the flexor retinaculum) supports the diagnosis, but a positive Phalen sign (flexing the wrist maximally and holding it in this position for 1 minute) is more sensitive.
6. Initial treatment is splinting in a volar splint with the wrist in neutral position and NSAIDs.
7. Surgery may be necessary; emergent hand specialty consult is required for median nerve motor deficits.

III. DUPUYTREN CONTRACTURE

A. Fibroblastic changes of the subcutaneous tissues of the palm and volar aspects of the fingers
B. Appears to have a genetic component
C. May lead to joint contractures with thickened, nodular palmar tissues

IV. GANGLION CYSTS

A. Cystic collection of synovial fluid within a joint or tendon sheath
B. Presents as a tender, cystic swelling over or near a tendon sheath
C. Management: pain control and NSAIDs
D. One-third of ganglion cysts resolve spontaneously, but persistent cysts may be referred to a hand specialist for cyst aspiration, corticosteroid injection, or surgical excision.

V. CHARCOT JOINT

A. Most common presentation is a painful, swollen ankle
B. Usual cause is diabetic peripheral neuropathy (not syphilis)
C. Radiograph of the ankle reveals classic "bag of bones" appearance.

VI. BACK PAIN

A. Differential diagnosis
1. Aortic aneurysm
2. Peptic ulcer
3. Pyelonephritis
4. Pancreatitis
5. Vertebral disc compression or fracture
6. Osteoporosis
7. Carcinoma (primary or metastatic)
8. Ankylosing spondylitis (associated with inflammatory bowel disease)

9. Infection
10. Osteomyelitis
11. Ectopic pregnancy
12. Strain or sprain
13. Pelvic inflammatory disease

B. General facts

1. Back pain is the most frequent (and the most costly) cause of workman's compensation payments.
2. Most back pain (including pain secondary to disc disease) is self-limited and resolves in ≤6–7 weeks, regardless of treatment.
3. Most back pain is benign (particularly in healthy, young- and middle-aged adults).
4. A serious cause of back pain is more likely in patients <20 years old (incidence of 10%) and in patients >55 years old (incidence of 20%).
5. 80% or more of low back pain occurs in the L4–S1 region; L5–S1 is the most frequent location.
6. When radiographs are indicated (which they are not in most cases), AP and lateral views alone are usually sufficient.
   a. Most healthy patients 18–55 years old with atraumatic, nonspecific, mechanical low back pain can be managed initially without radiographic evaluation.
   b. Lumbosacral films should be obtained as part of the initial evaluation in the following circumstances:
      (1) Age <18 or >55 years (higher incidence of serious causes)
      (2) Trauma
      (3) Persistent, unremitting back pain >4–6 weeks duration
      (4) Prior referral
      (5) Suspicion of serious underlying pathology based on the patient's:
         (a) Past medical history (cancer, IV drug abuse)
         (b) Vital signs (fever)
         (c) Atypical symptoms (night pain)

C. Lumbar disc syndromes

1. Impingement of a herniated disc on the anterior nerve root produces pain in a dermatomal pattern.
   L1: groin
   L2: upper thigh
   L3: mid thigh and medial knee
   L4: lower thigh, anterior knee, and medial foot
   L5: lateral lower leg, first interdigital web space, and dorsal foot
   S1: posterolateral calf and lateral foot
2. Clinical presentation: physical findings of nerve root involvement
   a. ↓ Knee jerk = L3 and L4
   b. ↓ Dorsiflexion of foot = L5
   c. ↓ Ankle jerk (↓ plantar flexion) and numbness of lateral foot = S1

3. **Cauda equina syndrome (neurosurgical emergency)**
   a. **A serious complication of lumbar disc disease**
   b. **Results from a massive midline lumbar disc herniation that compresses several nerve roots of the cauda equina**
   c. **Clinical presentation**
      (1) **Patients complain of lower back/leg pain and incontinence (or retention) of bowel and bladder. With progression, the patient also develops numbness of the feet and trouble walking.**
      (2) **Examination reveals loss of sensation in the "saddle" distribution and loss of anal tone (S3, S4, S5).**
   d. **Diagnostic evaluation: an MRI or emergency myelogram may be used to confirm the diagnosis and locate the disc level but should not delay treatment.**
   e. **Management: immediate neurosurgical consult for operative disc decompression; decompression must be performed quickly (within several hours) if permanent disability is to be avoided.**

D. Spondylolisthesis

1. Definition: forward displacement of a vertebral body on the one below (most commonly L5 on S1)
2. Clinical presentation
   a. Asymptomatic (only 25% of patients develop back or leg pain)
   b. Radiographic findings may be incidental and not necessarily the cause of the back/leg pain.
3. Diagnostic evaluation
   a. Best seen on the lateral view of the lumbar spine series
   b. The amount of forward displacement is graded; the higher the grade, the worse the slippage.
      (1) Grade I: up to 25%
      (2) Grade II: 25%–50%
      (3) Grade III: 50%–75%
      (4) Grade IV: 75%–100%
4. Management
   a. Outpatient protocol for patients with low back pain without neurologic findings ("lumbar strain")
      (1) Strict bed rest is no longer recommended for treatment of lumbar strain. Patients should be encouraged to do light activities, range of motion exercises, and stretches but should not be expected to do any prolonged walking, standing, twisting, or lifting of >15 pounds.
      (2) Analgesics (including NSAIDs)
      (3) Muscle relaxants
      (4) Follow-up in 7 days
      (5) Instructions to return immediately if problems develop (worsening pain, loss of motor strength, bowel or bladder incontinence)
   b. Inpatient management is indicated for patients with:
      (1) Cauda equina syndrome

(2) Severe or progressive neurologic deficit
(3) Multiple nerve root involvement
(4) Unmanageable pain
(5) Inadequate support system at home

E. Spinal infections

1. High-risk patients: IV drug users, immunocompromised patients, alcoholics, elderly, patients with prior blunt trauma or recent bacterial infection
2. **Epidural abscess**
   a. **True neurosurgical emergency**
   b. **20% of patients may have no comorbid illness.**
   c. **Most common bacterial cause is *S aureus*.**
   d. **Classic presentation is fever with neurologic symptoms, although 50% of patients may have neither finding.**
   e. **Risk of misdiagnosis is high on initial presentation.**
   f. **Mortality is as high as 23%.**
   g. **Treatment should include vancomycin in addition to broad-spectrum IV antibiotics. Consider *Pseudomonas* coverage for IV drug users.**
3. Spondylitis (osteomyelitis of the vertebral bone)
   a. Typically starts as subtle hematogenous seeding of the disc space, causing discitis. Contiguous spread then leads to vertebral endplate erosion.
   b. Most common pathogen is *S aureus*.
   c. May see enteric gram-negative bacilli or *Mycobacterium tuberculosis* (Pott disease)
   d. Typically presents with more indolent course and subjective fevers.

F. Spinal malignancy

1. Typically >50 years old
2. Presents with subacute or chronic back pain
3. Pain is worse at night.
4. Risk factors include a history of cancer, unexplained weight loss, persistent pain, and pain lasting >1 month.
5. Cancers that most commonly metastasize to bone include breast, lung, and prostate.

VII. HAND AND WRIST INFECTIONS

A. Paronychia

1. Infection of the lateral nail fold that sometimes extends to involve the proximal nail fold (eponychium)
2. Causative agents are usually *S aureus* or *Streptococcus*
3. Patients presenting early with cellulitis may respond to oral antibiotics (a penicillinase-resistant oral penicillin or a first-generation cephalosporin) and warm soaks alone.
4. If purulent material is present, the nail fold should be incised and drained, followed by daily warm soaks; use antibiotics only if a surrounding cellulitis is present.

5. If subungual pus is present, the adjacent portion of the nail should be removed.
6. Chronic paronychia may develop in immunocompromised patients. Consider treatment directed at atypical bacteria or fungal infections (*Candida albicans*).

B. Felon

1. **Infection of the pulp space of the fingertip**
2. **Causative agent is usually *S aureus*.**
3. **Management: incision and drainage at the point of maximum tenderness and fluctuance**
   a. **Make a central longitudinal incision starting 0.5 cm distal to the distal flexor crease (to avoid the flexor tendon sheath) or a unilateral longitudinal incision, and extend this incision into the pulp space. This approach is preferred because of fewer long-term complications.**
   b. **Pack the wound, splint the finger, and start the patient on an oral first-generation cephalosporin or antistaphylococcal antibiotic.**
   c. **Remove the packing in 48–72 hours, and start warm soaks.**
4. **Complications**
   a. **Flexor tenosynovitis and osteomyelitis**
   b. **Skin instability, loss of sensation, and a disrupted pad also may occur.**

C. Herpetic whitlow

1. **Viral infection of the distal finger**
2. **Caused by herpes simplex virus (type I typically in children, or type II typically in adults)**
3. **Typically occurs in healthcare providers with exposure to oral secretions and in patients with coexistent herpes infections.**
4. **Clinical presentation**
   a. **Localized burning, itching, and pain precede the development of the classic clear herpetic vesicles.**
   b. Typically, only one finger is involved.
5. **Diagnostic evaluation: Diagnosis can usually be made clinically, but if doubt remains or the presentation is atypical, it can be confirmed with Tzanck smear (reveals multinucleated giant cells) or viral culture.**
6. **Management**
   a. **Splinting, elevation, and analgesics for pain relief**
   b. **An oral antiviral agent effective against herpes simplex (eg, acyclovir, famciclovir) if patient is seen early in the course of the infection (or is immunocompromised)**
   c. **A dry dressing and instructions to prevent autoinoculation or transmission to others**
   d. **Surgical drainage is contraindicated; it can result in secondary infection and delayed healing.**

D. Human bite infections involving the metacarpophalangeal joint ("fight bites")

1. Can result in severe complications, including deep palmar space infections, functional loss, and amputation; these wounds must be aggressively treated and associated tendon injuries must be excluded.
2. Commonly result from punching someone in the mouth (initially denied by many patients)
3. A common mistake is to suture a human bite laceration over the metacarpophalangeal joint; these lacerations should never be closed primarily but allowed to heal by secondary intention.

4. Frequently found pathogens include anaerobes (especially *Eikenella corrodens* and anaerobic *Streptococcus*), *S aureus*, and *Neisseria* spp. Prophylaxis with amoxicillin-clavulanate may prevent this complication.
5. Management
   a. Consult with a hand surgeon.
   b. Obtain radiographs to exclude fractures and retained foreign bodies.
   c. Obtain aerobic and anaerobic wound cultures.
   d. Irrigate the wound with normal saline.
   e. Splint and elevate the hand.
   f. Admit the patient for IV antibiotics (ampicillin/sulbactam).

E. Pyogenic flexor tenosynovitis

1. **Typically results from a puncture wound**
2. **Causative agents are usually *S aureus* or *Streptococcus***
3. **Diagnosis is based on the presence of Kanavel four cardinal signs of flexor tenosynovitis**
   a. **Finger held in slight flexion**
   b. **Symmetric swelling of the finger**
   c. **Tenderness along the flexor tendon sheath**
   d. **Pain with passive extension of the finger**
4. **Management**
   a. **Hospitalization and emergent hand consult for surgical drainage**
   b. **IV antibiotics**
      (1) **If the tenosynovitis was caused by penetrating trauma → penicillinase-resistant antistaphylococcal penicillin or a first-generation cephalosporin**
      (2) **If there is no history (or evidence) of trauma in a sexually active adult, consider disseminated gonococcal infection and treat empirically with ceftriaxone until culture results are available.**
   c. **Elevation and splinting**
   d. **Tetanus prophylaxis as needed**

F. Deep palmar space infection

1. Characterized by swelling and tenderness localized to the palmar space
2. Management is incision and drainage in the operating room and IV antibiotics.

G. High-pressure injection injuries (true surgical emergencies)

1. **Mechanisms**
   a. **A 1–3 mm wound caused by a grease or paint gun is the hallmark of a high-pressure injection injury.**
   b. **Fusiform swelling and mild pain on initial presentation evolves to a swollen, pale, and severely painful digit hours later.**
   c. **The fluid frequently travels down the tendon sheath and damages the flexor tendon, with tenderness typically present along the course of injection.**
2. **Prognosis**
   a. **Poor, despite the deceptively normal appearance of the hand on initial presentation; amputation rates can range from 60% to 80%, but recent data demonstrate a 30% rate because of higher vigilance and early recognition of tissue injury.**

b. Factors affecting prognosis
   (1) Location of entry wound and the underlying anatomy affected
   (2) Physical and chemical qualities of the substance injected (viscosity, corrosiveness); substances that are low in viscosity and corrosive in nature (eg, paint solvents) produce the most damage.
   (3) Velocity of injection: the higher the velocity, the greater the penetration
   (4) Duration of exposure to the injected substance: the greater the duration of exposure, the worse the prognosis.

3. Management
   a. Obtain a radiograph to determine the degree of spread of material injected; it may be radiopaque.
   b. Splint and elevate the extremity.
   c. Administer a parenteral broad-spectrum antibiotic.
   d. Update tetanus prophylaxis as indicated.
   e. Provide oral or parenteral analgesia.
   f. Digital blocks are contraindicated → increased tissue pressure → vascular compromise
   g. Obtain immediate hand specialty consult for operative debridement.

VIII. COMPARTMENT SYNDROME (VOLKMANN ISCHEMIA) OF THE UPPER EXTREMITY

A. Usually involves the flexor (volar) compartment of the forearm

B. Arises from increased pressure in the muscle compartment of the limb (forearm) that compromises the circulation to the muscles and nerves of that compartment

C. Can be precipitated by an increase in compartment contents, a decrease in compartment size, or externally applied pressure; causes include:
   1. Supracondylar fracture of the elbow
   2. Fracture of the radius and ulna
   3. Forearm crush injury
   4. Extravasation of blood into the forearm
   5. Arterial injection of drugs
   6. Constrictive dressing or cast
   7. Burns

D. Clinical presentation
   1. Earliest finding is pain that is:
      a. Out of proportion to the injury
      b. Increased with passive stretching of the involved muscles
      c. Increased with active contraction of the involved muscles
   2. Paresthesia or hypesthesia of the nerves traversing the compartment (early finding)
   3. Paralysis or paresis of the involved muscles

4. Palpable tenseness and tenderness of the compartment
5. Pallor, cyanosis, or mottling of the skin (late finding)
6. Pulselessness or reduced distal pulses: rarely present (late finding)

E. Management

1. Remove constrictive dressings or casts (if present).
2. Obtain immediate orthopedic consult, and measure intracompartmental pressure.
3. Surgical decompression via fasciotomy is indicated if the pressure is >30 mmHg (normal pressure is <10 mmHg and typically near zero).
4. Do not elevate the limb; this does not significantly affect venous outflow and may further decrease perfusion.

F. Complications

1. If left untreated, compartment syndrome will result in Volkmann ischemic contracture.
2. Affected muscles become necrotic and are replaced with fibrotic tissue, thus producing a paralyzed and deformed arm.

IX. RHABDOMYOLYSIS

A. Dissolution or disintegration of striated muscle, which releases intracellular contents to circulation and extracellular fluid

B. Classic presentation is myalgias, weakness, red to brown (or "tea-colored") urine, and increased serum muscle enzymes

C. Can lead to life-threatening electrolyte imbalances, hepatic dysfunction, acute renal failure, compartment syndrome, multi-organ system failure, or death

D. Mechanism

1. Occurs when there is damage to the sarcolemma of the cell or depletion of ATP.
2. Intracellular calcium levels rise unregulated.
3. Calcium leads to persistent contraction and activation of calcium-dependent neutral proteases and phospholipids.
4. Electrolytes, myoglobin, and other intracellular contents are then released as the cell breaks down.
5. Excess myoglobin, combined with hypovolemia and acidic urine, can then lead to precipitation in the renal tubules, which can lead to acute renal failure.

E. General causes

1. Most common in the United States is prolonged muscle compression in the intoxicated patient or in a demented elderly patient after a fall
2. Metabolic myopathies
3. Drugs/toxins
4. Trauma/compression
5. Infections
6. Exertion

7. Electrolyte abnormalities
8. Electrical current
9. Hypoxia
10. Hyperthermia
11. Idiopathic

F. Management

1. Saline
   a. High volumes of normal saline
   b. Titrate to urine output of 200–300 mL/hour
2. Mannitol
   a. Controversial
   b. May provide a better prognosis in early acute renal failure by increasing urine flow, which may help prevent obstruction by myoglobin casts
3. Urine alkalinization
   a. Increases solubility of myoglobin
   b. Goal is urine pH of 6.5
   c. May use 2 ampules of sodium bicarbonate in 1 L of half-normal saline or 5% dextrose in water

G. Prognosis

1. Incidence of acute renal failure is unknown; may range from 13% to 50%.
2. Outcome is good provided there is no renal failure.
3. Incidence of renal failure is higher in patients who used illicit drugs or alcohol or have undergone trauma. Incidence is particularly higher in patients with more than one recognized cause.

MUSCULOSKELETAL DISORDERS (NONTRAUMATIC): PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A 58-year-old woman with a long history of rheumatoid arthritis presents with fever and left knee pain for the past 24 hours.

Physical examination: Her knee is swollen, warm, and tender.

Diagnostic evaluation: Laboratory evaluation reveals a leukocytosis and a markedly increased erythrocyte sedimentation rate. Aside from some soft-tissue swelling, radiographs are unremarkable. Arthrocentesis reveals purulent fluid (>100,000 WBCs with a predominance of PMNs), a low glucose, and gram-positive cocci in clusters on Gram stain.

What is the diagnosis?

Scenario B

Presentation: An obese 48-year-old man presents with left knee pain. He states that the pain developed over a period of a few hours and is so bad that even the weight of a bedsheet on his knee is too much to bear. He denies recent trauma and prior injury to the knee but admits to overindulging in rich food and alcohol the past couple of days. His past medical history is remarkable only for hypertension, for which he is being treated with a loop diuretic.

Physical examination: The patient's knee is erythematous, swollen, hot, and tender.

Presentation: Radiograph evaluation reveals only soft-tissue swelling. Synovial fluid analysis demonstrates 20,000 WBCs with a predominance of PMNs, a negative Gram stain, and needle-shaped crystals that are negatively birefringent.

What is the diagnosis?

Scenario C

Presentation: A 68-year-old woman presents with left knee pain for the past 2 days. She denies acute trauma and prior injury to the knee.

Physical examination: The patient's knee is erythematous, swollen, and tender.

Diagnostic evaluation: Radiographs reveal presence of calcium deposits. Arthrocentesis demonstrates 20,000 WBCs with a predominance of PMNs, a negative Gram stain, and rhomboid-shaped crystals that are positively birefringent.

What is the diagnosis?

Scenario D

Presentation: A 23-year-old IV heroin user presents after 2 days of back pain and fevers. She complains that she has tingling of the legs and cannot get comfortable regardless of how she moves or lays.

Physical examination: The patient is tachycardic and febrile on arrival and has diffuse lumbar back pain on percussion. Rectal tone is normal.

What is the diagnosis?

Scenario E

Presentation: A 67-year-old woman with a history of breast cancer presents with 2 months of constant back pain that is worse at night. There is no history of trauma or fevers, but she also reports a 12-pound weight loss.

Physical examination: There is midline tenderness over the upper lumbar spine without neurologic findings. Radiographs demonstrate a pathologic facture of the L1 vertebral body with loss of height.

What is the diagnosis?

Scenario F

Presentation: A 77-year-old woman with a history of dementia and alcohol abuse was found lying in the bathroom by her daughter after a fall the previous night. She complains of diffuse muscle aches, fatigue, generalized weakness, and dizziness.

Physical examination: The patient has diffuse muscle tenderness and appears dehydrated.

Diagnostic evaluation: A urine sample is dark brown. Laboratory studies show blood in the urine without RBCs present, and increased levels of potassium, BUN, creatinine, myoglobin, and total creatine kinase.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: septic arthritis

Scenario B

Diagnosis: gout

Scenario C

Diagnosis: pseudogout

Scenario D

Diagnosis: epidural abscess

Diagnostic evaluation: An MRI demonstrates a large epidural fluid collection in the L2–L5 space. C-reactive protein and WBC levels are increased.

Scenario E

Diagnosis: spinal malignancy

Scenario F

Diagnosis: rhabdomyolysis

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NEUROLOGIC DISORDERS: SELF-ASSESSMENT QUESTIONS

1. All of the following structures are supplied by the posterior circulation except:

   (a) Brain stem
   (b) Optic nerve and retina
   (c) Cerebellum
   (d) Thalamus

2. A 65-year-old patient presents with dizziness (vertigo), nausea and vomiting. On examination, he is noted to have lateralizing dysmetria and is unable to stand erect or ambulate without assistance. No other deficits are noted. The most likely diagnosis is:

   (a) Lacunar infarct
   (b) Vertebrobasilar artery occlusion
   (c) Middle cerebral artery occlusion
   (d) Cerebellar infarct

3. Transient global amnesia is characterized by all of the following except:

   (a) It generally occurs in patients >60 years old.
   (b) It is characterized by loss of recent memory and inability to record new memory.
   (c) Motor, sensory, and speech functions are interrupted during the event.
   (d) In general, patients have no recollection of events that occur during the episode.

4. The most appropriate therapy for refractory seizures secondary to isoniazid overdose is:

   (a) Thiamine
   (b) Pyridoxine
   (c) Magnesium sulfate
   (d) Lorazepam

5. All of the following statements regarding phenytoin are true except:

   (a) It is contraindicated in the presence of second- or third-degree heart block.
   (b) Signs of toxicity include nausea, vomiting, vertical nystagmus, ataxia, and lethargy.
   (c) Phenobarbital and chronic alcohol abuse increase serum phenytoin levels.
   (d) It should be administered at a rate of 25–50 mg/min.

6. Myopathies may be characterized by all of the following except:

   (a) Intact deep tendon reflexes
   (b) Diffuse or proximal weakness
   (c) Increased WBC count, sedimentation rate, and muscle enzymes
   (d) Profound sensory symptoms

7. Acute periodic paralysis generally occurs in males 7–21 years old and is associated with:

   (a) Hypokalemia
   (b) Normokalemia
   (c) Hyperkalemia
   (d) Hypocalcemia

8. Which of the following syndromes generally occurs in association with malignancy?

   (a) Guillain-Barré
   (b) Eaton-Lambert
   (c) Myasthenia gravis
   (d) Polymyositis

9. The most frequent initial symptoms of myasthenia gravis are:

   (a) Dysarthria and dysphagia
   (b) Muscle atrophy
   (c) Respiratory failure
   (d) Ptosis, diplopia, and blurred vision

10. All of the following statements regarding myasthenic crisis are accurate except:

    (a) The underlying problem is a functional excess of acetylcholine.
    (b) Patients can present with muscle weakness and respiratory compromise.
    (c) Administration of edrophonium would be expected to promote clinical improvement.
    (d) It typically occurs in undiagnosed/untreated patients and those with acute exacerbation of their disease process.

11. All of the following signs and symptoms suggest peripheral vertigo except:

    (a) Presence of hearing loss and tinnitus
    (b) Vertical nystagmus
    (c) Spontaneous nystagmus that is suppressed by visual fixation
    (d) Absence of associated neurologic abnormalities

12. The corneal reflex assesses which cranial nerves?

    (a) V and VII
    (b) III and V
    (c) III and VII
    (d) II and VII

13. All of the following statements regarding subarachnoid hemorrhage are accurate except:

    (a) The sensitivity of CT scanning in detecting subarachnoid hemorrhage is about 80%–95%.
    (b) ECG changes that resemble those produced by cardiac ischemia can occur.
    (c) Lumbar puncture is the initial procedure of choice for detecting subarachnoid hemorrhage.
    (d) Patients classically present with the worst headache of their lives, are confused and may have other nonfocal neurologic signs.

14. A 35-year-old man presents with a severe boring, unilateral headache that woke him from sleep. He had the same type of headache the night before. Physical examination reveals lacrimation, flushing, and rhinorrhea on the affected side. His neurologic examination is otherwise normal. The most effective acute therapy is:

    (a) Cyclic antidepressants
    (b) Calcium channel blockers
    (c) 100% oxygen
    (d) Lithium carbonate

15. The most appropriate empiric antibiotic therapy for a neonate with meningitis is:

    (a) Penicillin G
    (b) Ampicillin and cefotaxime
    (c) Cefuroxime
    (d) Ceftriaxone

16. Common causes of meningitis in normal adults 18–50 years old are:

    (a) *Streptococcus pneumoniae* and *Niesseria meningitidis*
    (b) *Listeria monocytogenes* and gram-negative bacilli
    (c) *Haemophilus influenzae* and *S pneumoniae*
    (d) *S pneumoniae* and Group B streptococci

ANSWERS

1. b	5. c	9. d	13. c
2. d	6. d	10. a	14. c
3. c	7. a	11. b	15. b
4. b	8. b	12. a	16. a

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. STROKE

A. Anatomy of cerebral blood flow

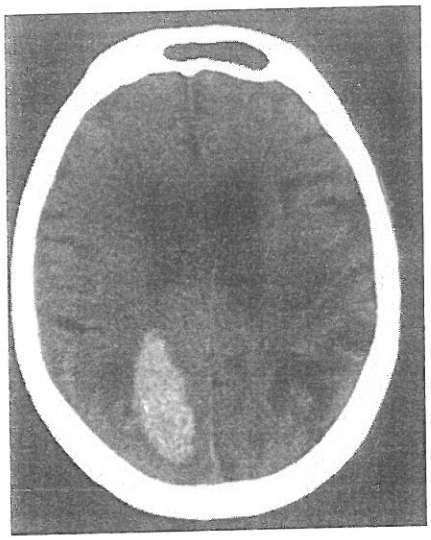
1. Anterior circulation: 80% of cerebral blood flow originates from the carotid arteries, which supply the:
   a. Frontoparietal lobes
   b. Anterior aspect of temporal lobes
   c. Optic nerve and retina
   d. Many deep gray matter structures
2. Posterior circulation: 20% of cerebral blood flow originates from the vertebrobasilar arteries, which supply the:
   a. Medial aspect of temporal lobe
   b. Visual occipital cortex
   c. Thalamus
   d. Brainstem
   e. Upper spinal cord
   f. Cerebellum
   g. Auditory and vestibular functions of the ear
3. Circle of Willis: provides a connection between the anterior and posterior circulations; its integrity determines the clinical outcome of thromboembolic strokes because of the collateral flow provided.

B. Pathophysiology of cerebral ischemia and infarction

1. Ischemic strokes (may be caused by cerebrovascular thrombosis, emboli, or structural occlusion of a vessel)
   a. Cerebral in situ vascular occlusion (thrombosis) results from one of the following:
      (1) Atherosclerosis (most common cause in patients >50 years old)
         (a) Atherothrombotic infarcts are associated with plaques in the carotid and vertebrobasilar arteries or the middle cerebral artery.
         (b) Cerebral vessel branch points are common areas for thrombotic occlusion, because clot formation is prone to develop in areas of turbulent blood flow.
         (c) Patients of African-American descent and those with diabetes have a particularly high risk of intracranial thrombosis.
      (2) Hypercoagulable states (common cause in patients <50 years old)
         (a) Antiphospholipid antibody
         (b) Factor V Leiden mutation
         (c) Protein C or S deficiency
         (d) Sickle cell disease
         (e) Thrombocytosis
      (3) Vasculitis (common cause in patients <50 years old)
      (4) Mycotic aneurysms
      (5) Polycythemia vera
      (6) Spontaneous or post-traumatic carotid or vertebral artery dissection

b. Cerebral emboli result from one of the following:
   (1) Plaques from large neck vessels or the proximal aorta (common source)
   (2) Mural thrombi from the heart (associated with atrial fibrillation, recent MI, prosthetic valves, or native valvular heart disease) (common source)
   (3) Dilated cardiomyopathy
   (4) Endocarditis (embolism of bacterial/fungal vegetations)
   (5) Thoracic or dysbaric injury (air emboli)

2. Hemorrhagic strokes result from one of the following:



Courtesy of Michael Abraham, MD
CT scan showing right-sided temporoparietal intraparenchymal hemorrhage

a. Spontaneous rupture of a congenital or berry aneurysm (subarachnoid hemorrhage)
b. Spontaneous rupture of an AV malformation (less common cause of a subarachnoid hemorrhage)
c. Spontaneous bleeding from arterioles (accounts for most cerebral hemorrhages) and are secondary to one of the following:
   (1) Chronic hypertension (arteriolar injury)
   (2) Ruptured mycotic aneurysm
   (3) Neoplasm (especially breast cancer, melanoma)
   (4) Thrombocytopenia
d. Amyloid angiopathy
   (1) Common cause of intracerebral hemorrhage in the elderly
   (2) CT → distinctive location of the hemorrhage at the junction of gray and white matter
e. Hemorrhagic transformation of an embolic (ischemic) stroke is:
   (1) Characterized by abrupt progression of neurologic dysfunction after a stroke *and*
   (2) Associated with fragmentation of an occluding embolus or administration of thrombolytics (there does not appear to be an increased risk of hemorrhage in patients on chronic ASA therapy).
   (3) Not always evident on CT; recent studies suggest that gradient recall echo MRI may be a better choice.

C. Incidence of stroke

1. Patients with a stroke frequently have a history of one or more of the following:
   a. Transient ischemic attack (TIA)—30%–35% of patients will have a stroke within 5 years, the highest incidence occurring within the first month and the earliest incidence within 2 days
   b. Hypertension (most common)
   c. Atherosclerosis
   d. Cardiac disease
   e. Migraine headaches
   f. Claudication
   g. Diabetes
   h. Hyperlipidemia
   i. Smoking
   j. Thrombocytosis
   k. Oral contraceptive use (high-estrogen formulas)
   l. Erythrocytosis
2. A carotid bruit
   a. Absence of a bruit does not exclude the presence of stenosis.
   b. Presence/absence of a bruit has no predictive value regarding the likelihood of stroke related to carotid artery disease; however, if >70% carotid stenosis is confirmed radiographically, there is a 26% risk of stroke within 2 years in patients who have had a TIA.

D. Differential diagnosis of stroke (aka mimics)

1. **Seizure**
   a. **Although embolic strokes may present with seizures, a transient postictal neurologic deficit (such as Todd paralysis) is far more common.**
   b. **Patients with other intracranial pathology (post-traumatic hematoma formation, tumor, abscess) may also present with seizures and neurologic deficits. Patients with new focal deficits in the setting of seizure should undergo emergent CT scanning to look for structural CNS lesions. (*ACEP Clinical Policy:* Initial Approach to Patients Presenting with a Chief Complaint of Seizure Who Are Not in Status Epilepticus)**
2. **Migraine**
   a. **The aura preceding migraine headache may include focal neurologic deficits (eg, weakness, numbness, aphasia); a previous history of focal symptoms associated with migraine is required to make this diagnosis. Transient ischemia associated with migraine may also result in a stroke.**
   b. **MRI with diffusion-weighted imaging may help distinguish migraine from cerebral infarction.**
3. **Trauma to the middle meningeal artery (epidural or extradural hematomas)**
4. **Traumatic or spontaneous venous bleeding (subdural hematoma)**
5. **Toxic/metabolic disorder (those listed below may cause focal neurologic deficits and, if present, may exclude a stroke)**
   a. **Toxic exposure (some drugs, poisons)**
   b. **Hypo/hyperglycemia**

c. **Hypo/hypernatremia**
d. **Hepatic encephalopathy**

E. Ischemic stroke syndromes
1. A TIA should be considered a warning sign of an impending thrombotic or embolic syndrome. It is a "red flag," heralding stroke in evolution.
2. Differentiating thrombotic and embolic strokes
   a. Thrombotic syndromes are often characterized by a stuttering onset of symptoms. Some patients wake up with neurologic disability.
   b. Embolic syndromes are characterized by abrupt onset of symptoms with maximal deficit that improves slowly as the embolus breaks up and moves peripherally. Embolic strokes are more likely to occur when the patient is active.
3. Occlusive stroke syndromes may be embolic or thrombotic.
   a. Middle cerebral artery
      (1) Most common site of intracranial cerebral artery thrombosis
      (2) Clinical presentation (frequently incomplete)
         (a) Contralateral hemiplegia, hemisensory loss, and homonymous hemianopsia
         (b) Upper extremity deficit that is more severe than lower extremity deficit
         (c) Aphasia (if the dominant hemisphere is involved)
         (d) Constructional apraxia and agnosia (if the nondominant hemisphere is involved)
         (e) Gaze preference in the direction of the lesion
   b. Anterior cerebral artery
      (1) Clinical presentation
         (a) Contralateral leg, foot, and arm paralysis
         (b) Sensorimotor deficit greater in the lower (rather than the upper) extremity
         (c) Loss of frontal lobe control
            i. Abulia (difficulty in responding to questions because of slowed mentation)
            ii. Gait apraxia (clumsiness)
            iii. Incontinence
            iv. Primitive grasp and suck reflexes
   c. Posterior cerebral artery
      (1) Affects blood supply to the occipital cortex and branches that supply upper midbrain structures
      (2) Clinical presentation
         (a) Contralateral homonymous hemianopsia (the patient may be unaware of this before clinical evaluation), hemiparesis, and hemisensory loss
         (b) Memory loss
         (c) Ipsilateral cranial III nerve palsy (usually pupil-sparing)
   d. Vertebrobasilar artery (not all findings are present)
      (1) The vertebrobasilar arteries are affected by both atherosclerotic narrowing and embolic events that may result in:
         (a) Cerebellar and cranial nerve deficits
         (b) Simultaneous deficits on both sides of the body
      (2) Clinical presentation

(a) Pain and temperature deficits on one side of the face and on opposite side of the body
(b) Ipsilateral cranial nerve palsies
(c) Cerebellar signs
i. Those that suggest labyrinthitis
- Vertigo
- Severe ataxia
- Nausea/vomiting

ii. Those that, along with cranial nerve palsies, are not present in patients with labyrinthitis
- Vertical or bidirectional nystagmus
- Ipsilateral dysmetria
- Dysdiakokinesis

(d) Contralateral hemiplegia
(e) Contralateral sensory deficits

(3) Wallenberg syndrome (lateral medullary plate syndrome)
(a) Occurs when occlusion of the proximal vertebral artery causes infarction of the lateral medulla
(b) Clinical presentation
i. Vertigo
ii. Dysphagia
iii. Horner syndrome
iv. Facial pain/numbness
v. Ipsilateral clumsiness
vi. Contralateral cool extremity (distally)

e. Basilar artery
(1) Characterized by severe bilateral signs
(2) Clinical presentation
(a) Quadriplegia
(b) "Locked-in syndrome" (loss of all motor function except upward gaze)
(c) Coma

f. Cerebellar infarcts
(1) Clinical presentation
(a) Ataxia
(b) Dizziness (vertigo)
(c) Nausea/vomiting
(d) Nystagmus (vertical or bidirectional)
(e) Lateralizing dysmetria (finger-nose-finger, heel-shin, past-pointing)
(f) Dysdiadokokinesis (rapid alternating movements)

g. Lacunar infarcts
(1) These small cystic infarcts are usually caused by a hypertensive vasculopathy (lipohyalinosis) but may occur in diabetic patients and can affect both the anterior

and posterior cerebral vessels. Lacunar strokes involve penetrating cerebral arterial vessels lying deep in the gray matter (internal capsule) or brainstem.

(2) There are four specific lacunar infarct states

(a) The "dysarthria-clumsy hand syndrome" (midpons)

(b) Pure sensory syndrome (thalamus)

(c) Leg paresis and ataxia (pons or internal capsule)

(d) Pure motor hemiplegia (pons or internal capsule): most common

h. Transient global amnesia

(1) Associated with ischemia to the temporal lobes or thalamus

(2) Generally occurs in men >60 years old, lasts from 30 minutes to 36 hours, and then resolves completely; has also been reported in association with therapeutic use of benzodiazepines (especially triazolam).

(3) Clinical presentation

(a) Sudden loss of recent memory and inability to record new memories; the patient usually has no recollection of events that occurred during the episode.

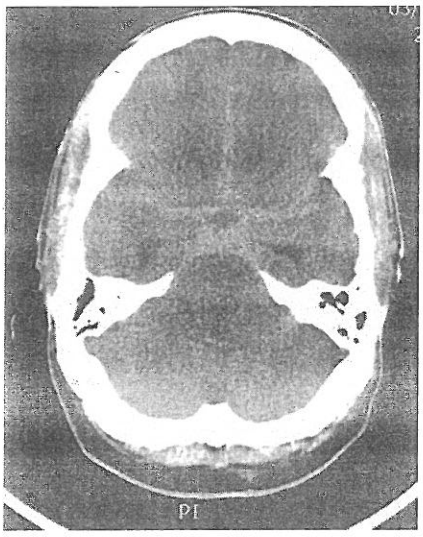
(b) Preservation of long-term past memory and self-identity

(c) Preservation of motor, sensory, and speech functions

(d) The ability to carry out complex tasks (drive a car, give a speech) during the episode

F. Hemorrhagic syndromes

1. Characterized by abrupt onset of symptoms (usually headache, vomiting) and rapid progression of neurologic deficits. The patient may be lethargic or exhibit decreased mentation by the time he or she arrives in the emergency department. However, a more subtle presentation (not marked by a rapid clinical deterioration) is also common. Initial blood pressure is usually high in patients with significant hemorrhage.
2. Subarachnoid hemorrhage (SAH): nontraumatic



Courtesy of Michael Abraham, MD

Classic CT scan of SAH

a. Highest incidence in women is in those 35–65 years old and in men in those <40 years old
b. Associated with coarctation of the aorta, polycystic kidneys, Marfan syndrome, Ehlers-Danlos syndrome, and fibromuscular dysplasia
c. Spontaneous SAH generally results from rupture of a saccular (berry) aneurysm.
d. Clinical presentation
   (1) Patient complains of sudden onset of an "unusual headache," which may improve spontaneously or with non-narcotic analgesics.
   (2) Nuchal rigidity, photophobia, and vomiting may or may not be present.
   (3) Funduscopic examination may reveal preretinal hemorrhage.
   (4) Focal neurologic findings are generally absent, and the mental status examination is often normal.
e. Diagnostic evaluation
   (1) CT of the brain is most sensitive in the first 6 hours after the bleed and is useful in differentiating SAH from other causes of severe headache (migraine).
   (2) If the scan is negative, a lumbar puncture should be done, because CT is ~96% sensitive.
   (3) MRI may be as accurate as CT in patients with focal signs or symptoms.
   (4) Lumbar puncture findings suggestive of SAH
      (a) Grossly bloody fluid
      (b) CSF does not progressively clear (unlike in a traumatic tap, in which the first tube of CSF taken immediately after the lumbar puncture shows higher RBC counts than the last tube)
      (c) Xanthochromia (takes up to 12 hours to develop and lasts 2 weeks): due to the degradation of RBC in the CSF
      (d) Normal (or ambiguous) CSF findings and a nondiagnostic CT in a patient with a high pretest risk of SAH do not exclude an unruptured (yet symptomatic) aneurysm. MRI or angiography is indicated in these patients.
   (5) Angiography is not routinely indicated to exclude SAH for patients in whom the CT and lumbar puncture are both normal. (*ACEP Clinical Policy:* Critical Issues in the Evaluation and Management of Patients Presenting to the Emergency Department with Acute Headache)

3. Hypertensive intracerebral hemorrhage
   a. Putamen hemorrhage (most common)
      (1) Similar in presentation to middle cerebral artery syndrome
         (a) Contralateral hemiplegia, hemianesthesia, and homonymous hemianopsia; gaze palsy may be present.
         (b) Aphasia (if the dominant hemisphere is involved) or hemineglect (if the nondominant hemisphere is involved)
      (2) Distinguishing feature: greater depression of consciousness with putamen hemorrhage than with middle cerebral artery occlusion
   b. Cerebellar hemorrhage
      (1) Sudden onset of ataxia, dizziness, occipital headache, repeated vomiting, and loss of ability to stand or walk is typical.
      (2) If diagnosed early, this condition is treatable with surgical decompression and hematoma evacuation.

c. Thalamic hemorrhage
   (1) Contralateral hemiparesis and hemianesthesia
   (2) Sensory loss > motor loss
   (3) Restricted upward gaze or abnormal deviation of the eyes without a visual field defect (occasionally)
   (4) Coma (may resolve if associated with focal depression of reticular activating system)

d. Pontine hemorrhage
   (1) Severe occipital headache with rapid progression to coma (associated with poor prognosis)
   (2) Decerebrate posturing
   (3) Hyperventilation
   (4) Pinpoint pontine pupils ("3 P's"); other causes of pinpoint pupils:
      (a) Narcotic overdose (associated with hypoventilation)
      (b) Cholinesterase inhibitor poisoning (pesticides)
      (c) Pilocarpine eye drops or systemic atropine
      (d) Clonidine overdose
   (5) Absence of oculovestibular reflexes on caloric testing and doll's head maneuver

G. Treatment of stroke

1. Intubation should be considered in any stroke patient with a depressed level of consciousness.
2. **Hypertension is commonly associated with acute stroke. Should it be treated?**
   a. **The general consensus in the neurology literature is not to treat hypertension in patients with acute thrombotic stroke unless they are candidates for thrombolysis and blood pressure is >185 mmHg systolic or >110 mmHg diastolic; this is because of the following reasons:**
      (1) **Patients with chronic hypertension may suffer greater ischemia when the pressure is lowered.**
      (2) **Perfusion of the ischemic but still viable (ischemic penumbra) tissue around the stroke area may require a higher blood pressure.**
   b. **Treatment of patients with hemorrhagic stroke is less clear, but the neurosurgery literature suggests that significant increases in blood pressure be treated.**
      (1) **Labetalol is the drug of choice that preserves normal cerebral autoregulation.**
      (2) **Nitroprusside is not a good choice, because vasodilators may cause "steal" effects (increasing ischemia in the injured tissue) and also increase intracranial pressure.**
      (3) **Nicardipine is also a good choice, because it secondarily prevents cerebral vasospasm and rebleeding rates in hemorrhagic strokes.**
3. If the patient is not a candidate for thrombolysis, ASA (160–325 mg) should be given in the setting of acute ischemic stroke (within 48 hours). Multiple studies support this practice.
4. Heparin therapy has not been shown to reduce stroke-related mortality or disability in the setting of acute stroke (although it is highly effective in the prevention of cardioembolic strokes).
5. Guidelines for the use of thrombolytics in patients with acute stroke have been defined by the American Academy of Neurology, the American Heart Association, and the American Surgical Association (2013).

a. Diagnosis of ischemic stroke causing measurable neurologic deficit
b. Neurologic signs not rapidly resolving
c. Onset of symptoms <3 hours before beginning treatment
d. No head trauma or prior stroke in past 3 months
e. No MI in past 3 months
f. No GI, trauma, or bleeding episodes in recent history
g. Systolic blood pressure <185 mmHg, diastolic blood pressure <110 mmHg
h. No evidence of acute trauma or bleeding
i. Platelet count >100,000/mm^3
j. Blood glucose >50 mg/dL (2.7 mmol)
k. CT scan does not show evidence of multilobar infarction (hypodensity over one-third hemisphere)
l. Time of administration of tissue plasminogen activator can be extended to 4.5 hours if patient is <80 years old, not taking anticoagulants, does not have a history of diabetes or stroke, or has an NIHSS score >25.

6. Patients with significant edema (or those who have a very large infarct) have a greater risk of bleeding, but they are not excluded in current guidelines. (Ideally, thrombolytic therapy in the stroke setting should use a team approach that includes expert interpretation of the CT scan to exclude the presence of hemorrhage. Admission to a specialized stroke unit also maximizes recovery of stroke patients.)

II. SEIZURES

A. General information

1. Incidence: 1%–2% of the general population has recurrent seizures.
2. Etiology
   a. Primary (idiopathic) epilepsy: onset typically at 10–20 years of age
   b. Secondary (symptomatic) epilepsy is precipitated by one of the following:
      (1) Intracranial pathology
         (a) Mass (tumor, cyst, old scar)
         (b) Abscess
         (c) Trauma
         (d) Vascular lesion (AV malformation, aneurysm, stroke)
      (2) Extracranial pathology
         (a) Toxic (eg, alcohol withdrawal, cocaine, tricyclic antidepressant overdose)
         (b) Uremic or hypertensive encephalopathy
         (c) Eclampsia
         (d) Endocrine/electrolyte abnormalities, hypoglycemia

B. Types

1. Partial seizures
   a. Simple partial (focal) seizures → motor, sensory, autonomic or psychic onset depending on where the electrical discharge is coming from; no loss of consciousness

b. Complex partial seizures (temporal lobe or psychomotor) → motor, sensory, autonomic, or psychic onset (may mimic functional psychosis) associated with impaired consciousness, automatisms (lip smacking, picking at clothes), and postictal confusion

c. Secondary generalized partial seizures (tonic-clonic or grand mal) → motor, sensory, autonomic or psychic onset followed by a generalized seizure, coma, and slow return to consciousness; tongue biting or incontinence may occur.

2. Generalized seizures

a. Nonconvulsive: absence (petit mal) seizures → brief (~10 seconds) alteration in level of consciousness (eg, cessation of activity associated with staring in a school-aged child); motor tone may increase or decrease with mild clonic movements or automatisms, which may be precipitated by hyperventilation.

b. Convulsive: tonic-clonic, grand mal

3. Todd paralysis: a transient focal paralysis after a seizure that usually lasts 1–2 hours but can last 1–2 days

C. Differential diagnosis

1. Myoclonus (may be associated with a multitude of disorders that include: Creutzfeldt-Jakob disease, uremia, or lithium toxicity)
2. Narcolepsy/cataplexy
3. Tetanus
4. Syncope
5. Psychogenic seizures (pseudoseizures)
6. Strychnine poisoning (patients remain alert)

Table 18: Historical Clues that Distinguish a Generalized Seizure from Syncope

Generalized Seizure	Syncope
Occurs without warning	Prodrome of darkening vision, nausea
Tonic-clonic movements	May have brief clonic movements
Prolonged postictal period	No postictal period after return of normal cerebral blood flow

D. Diagnostic evaluation for a first seizure, questionable seizure, unusual seizure, or seizure that is complicated by injury, illness, or substance abuse

1. The only routine tests that have a reasonable yield in the evaluation of a first seizure are a serum glucose and sodium; so-called baseline studies (CBC, urinalysis, electrolytes other than sodium, BUN/creatinine, etc) are only indicated by clinical assessment, so a good history and physical examination are essential.
2. Other tests that may be useful (again, based on history and physical examination)

a. Lumbar puncture

b. Blood alcohol, toxicology screen

c. Anticonvulsant levels (in patients taking them)

d. Chest radiograph (if tumor or aspiration is suspected)

3. Indications for neuroimaging

a. Emergent CT or MRI

(1) Postictal patients with:

(a) Status epilepticus
(b) Recent head injury
(c) Prolonged postictal state
(d) Severe headache
(e) New focal deficit

(2) Patients with new-onset seizures whose follow-up for routine CT cannot be assured. (*ACEP Clinical Policy:* Initial Approach to Patients Presenting with a Chief Complaint of Seizure Who Are Not in Status Epilepticus)

b. Urgent CT (performed during emergency department evaluation)
(1) Suspected CNS lesion
(2) New seizure (in patients at risk of neurocysticercosis or HIV, and those with a history of malignancy)

E. Status epilepticus

1. Definition: seizures that persist for ≥5 minutes or repetitive seizures without a lucid interval; permanent neuronal damage may result within 1 hour in patients with uncontrolled seizures; mortality rate is as high as 30%.
2. **Treatment (adults)**
   a. **Intubation may be indicated for hypoxia or risk of aspiration; 100% oxygen and continuous pulse oximetry are indicated. (If a long-acting paralytic agent is used for airway management, EEG monitoring is indicated, because paralysis masks seizure activity.)**
   b. **Large-bore IV line using a non–dextrose-containing solution followed by:**
      (1) **Diazepam 2 mg/min IV up to 0.1 mg/kg (preferred by some clinicians because of duration of action); alternatively, lorazepam can also be administered and has a faster onset of action.**
      (2) **Phenytoin 18–20 mg/kg IV at a rate of 25–50 mg/min, or fosphenytoin 18–20 phenytoin equivalents (PEs/kg) at 100–150 PEs/min. Phenytoin cannot be given IM because it is erratically absorbed. Fosphenytoin, a prodrug that is rapidly metabolized to phenytoin, has two advantages: a more rapid rate of administration IV and good absorption after IM administration. Phenytoin is not effective for seizures related to toxins (eg, theophylline, cocaine) or for ethanol withdrawal.**
      (3) **If no response, administer phenobarbital 8–20 mg/kg at a rate of 60 mg/min.**
      (4) **Thiamine 100 mg IV and glucose 25–50 g 50% solution (if bedside testing reveals hypoglycemia or if alcoholism or malnutrition is suspected)**
      (5) **Magnesium sulfate 20 mL 10% solution IV if patient eclamptic (pregnant or postpartum), alcoholic, or malnourished**
      (6) **Pyridoxine (5% or 10% solution) 5 g IV (if isoniazid overdose suspected)**
      (7) **If there is still no response, consider one of the following:**
         (a) **Barbiturate coma regimen (pentobarbital 5 mg/kg at 25 mg/min, then titrate to EEG), *or***
         (b) **Diazepam infusion (100 mg in 500 mL D5W) run at 40 mL/hr (8–10 mg/hr)**
3. **Treatment (children)**
   a. **Airway management (intubation and oxygen)**
   b. **IV line followed by:**

(1) **Rapid glucose determination → glucose 2–4 mL/kg 25% solution as needed (D10 in neonates)**

(2) **Pyridoxine 50–100 mg slowly IV (for neonates)**

(3) **Calcium gluconate 4 mL/kg IV (for neonates)**

(4) **Lorazepam 0.05–0.1 mg/kg at a rate of 2 mg/min (if an IV line cannot be established quickly, diazepam may be given rectally at 0.3–0.5 mg/kg)**

(5) **If no response → fosphenytoin 15–20 mg/kg IV at a rate ≤40 mg/min**

(6) **If no response → phenobarbital 15–20 mg/kg IV at 25–30 mg/min**

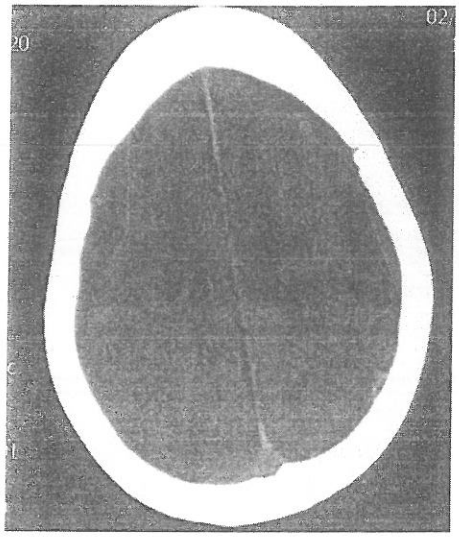
4. Additional facts regarding drug therapy
   a. Both phenytoin and diazepam are erratically absorbed and should not be given IM. Lorazepam and fosphenytoin are rapidly absorbed when given IM.
   b. Rapid infusion of phenytoin has been associated with hypotension, dysrhythmias, and heart block. This is an effect of the propylene glycol diluent and not of the drug itself. Even very high levels of phenytoin are not associated with cardiac dysrhythmias.
   c. Full loading doses of phenobarbital are associated with respiratory depression. The need to administer a full loading dose for control of status epilepticus is a clear indication for intubation.
   d. Phenobarbital and chronic alcohol abuse →↑ serum phenytoin levels
   e. Chloramphenicol, isoniazid, warfarin, sulfonamides, cimetidine, and omeprazole → ↑ serum phenytoin levels (Sudden withdrawal from isoniazid in a patient on phenytoin may result in ↓ serum phenytoin levels and subsequent seizures.)
   f. Phenytoin toxicity (therapeutic serum level: 10–20 mcg/mL)
      (1) Lateral gaze nystagmus (20 mcg/mL)
      (2) Increasing ataxia, prominent nystagmus that may be vertical (>30 mcg/mL)

III. COMA (PATIENT IS UNRESPONSIVE TO VERBAL AND PAINFUL STIMULI)

A. Etiology: mnemonic "TIPS AEIOU"

Trauma, **T**emperature
Infection
Porphyria, **P**sychiatric disorder
Space-occupying lesion, **S**ubarachnoid hemorrhage, **S**troke, **S**hock
Alcohol (drugs and toxins)
Endocrine, **E**xocrine and **E**lectrolyte disorders
Insulin
Oxygen, **O**piates
Uremia

1. Trauma (primarily head injury)
   a. Subdural hematoma



Courtesy of Michael Abraham, MD
CT scan showing bilateral subdural hemorrhage.

   b. Epidural hematoma
2. Temperature
   a. Hypothermia: causes coma when temperature is <82.4°F (28°C) and may be associated with a toxin, myxedema, or hypopituitarism
   b. Hyperthermia: causes coma when temperature is >107.6°F (42°C)
3. Infection
   a. Meningitis
   b. Encephalitis
   c. Sepsis
4. Endocrine, exocrine, and electrolyte disorders
   a. Hypo/hyperglycemia
   b. Hypo/hypernatremia
   c. Hypo/hypercalcemia
   d. Hypo/hyperthyroidism
   e. Hepatic encephalopathy (↑ NH_3)

B. Physical examination

1. Always check and stabilize the ABCs before any detailed evaluation.
2. Because there is frequently little or no known history in these patients, perform a thorough physical examination, including completely exposing the patient.
3. Smell the breath.
   a. Fruity (diabetic ketoacidosis)
   b. Feculent (bowel obstruction)
   c. Putrid (anaerobic infection)
   d. Garlic (organophosphate insecticide)
   e. Bitter almonds (cyanide): only 60%–80% of the population can detect this.
   f. Alcohols (ethanol, isopropyl alcohol); methanol and ethylene glycol are odorless.
   g. Ammonia (uremia)

h. Wintergreen (methyl salicylate)
i. Fetor hepaticus: a musty smell (liver failure)

4. Observe the respiratory pattern; it may have diagnostic value.
   a. Cheyne-Stokes: periodic, regularly increasing breaths in a crescendo pattern, followed by decrescendo breathing and ending with short periods of apnea that are due to dysfunction (structural or metabolic) of both cerebral hemispheres or upper brainstem
   b. Hyperventilation: causes include:
      (1) Hypoxia
      (2) Metabolic acidosis
      (3) Salicylate overdose
      (4) Lesions of the midbrain
      (5) Hepatic coma
      (6) ↑ Intracranial pressure (ICP)
   c. Apneustic breathing: an end-inspiratory pause that occurs in association with lesions of the pons in the area of cranial nerve V
   d. Cluster breathing: short bursts of breathing that are associated with lower pontine lesions
   e. Ataxic breathing: an irregular breathing pattern that precedes death and is associated with lesions of the medulla
5. Look for any abnormal posturing. Both decorticate and decerebrate posturing may be seen in patients with hypoglycemia or severe hepatic encephalopathy.
   a. Decorticate posturing
      (1) Flexion of the upper extremities and extension of the lower extremities
      (2) Suggests a lesion in the upper midbrain or internal capsule
   b. Decerebrate posturing: extension of all extremities; suggests a deeper, more severe dysfunction
      (1) Severe midbrain lesions
      (2) Posterior fossa lesions that compress or partially damage the midbrain and pons
      (3) Severe metabolic disorders (hepatic coma, drug overdose) that depress the function of the deep diencephalon and forebrain
      (4) Bilateral diffuse hemispheric abnormalities (postanoxic cerebral demyelination)
6. Look at the skin.
   a. Needle tracks (IV drug abuse)
   b. Cyanosis (hypoxia, hemoglobinopathy)
   c. Pallor (anemia)
   d. Cherry red (carbon monoxide)
   e. Petechial or purpuric rash (meningococcemia, Rocky Mountain spotted fever)
   f. Diaphoresis (hypoglycemia, cholinergic/salicylate poisoning)
   g. Pressure bullae (barbiturates, carbon monoxide, meprobamate)
   h. Ecchymoses at common sites of insulin injection (hypoglycemia)
7. Look for ocular signs.
   a. Pupillary reactivity (the most important physical sign in distinguishing metabolic from structural causes of coma)
      (1) Reactive (but sluggish) pupils are characteristic of a metabolic or toxic disorder.

(2) Nonreactive pupils are associated with a structural lesion with the following exceptions:
  (a) Anoxia (fixed and dilated)
  (b) Atropine and scopolamine (fixed and dilated)
  (c) Hypothermia (may be fixed)

b. Anisocoria (unequal pupils ≥1 mm difference)
  (1) Found in 10%–20% of the normal population
  (2) Other causes
    (a) Local eye trauma (traumatic mydriasis)
    (b) Instillation of a cycloplegic agent in one eye
    (c) Prior eye surgery
    (d) Compression of cranial nerve III by an enlarging aneurysm
    (e) Uncal herniation

c. Other pupillary abnormalities
  (1) Pupil initially constricts to direct light during the swinging light test but dilates paradoxically as the light swings back to it, because it perceives a less intense stimulus than the normal eye perceived → Marcus-Gunn pupil (retina/optic nerve pathology [unilateral])
  (2) Large, irregular pupil constricts very slowly to direct light → Adie's pupil (diabetic neuropathy, alcoholism)
  (3) Dilated pupil unreactive to light (most commonly cranial nerve III [or cycloplegic])
  (4) Small, unilateral pupil reacts to light → Horner syndrome (interruption of the sympathetic nerve supply, usually in the neck)
  (5) Small, bilateral pupils unreactive to light but reactive to accommodation → Argyll-Robertson pupil (suggests neurosyphilis)

d. Fundi
  (1) Spontaneous venous pulsations (normal ICP)
  (2) Papilledema (↑ ICP, chronic)
  (3) Subhyaloid hemorrhages

e. Ocular reflexes
  (1) Corneal reflex tests cranial nerves V (sensory) and VII (motor); absent with lesions in the posterior fossa or brainstem.
  (2) Oculocephalic reflex (doll's eyes): if the cervical spine has been cleared, turn the head in either the horizontal or vertical plane; a normal response (intact brainstem in a comatose patient) is conjugate deviation of the eyes in a direction opposite to the head rotation.
  (3) Oculovestibular reflex (cold calorics): Elevate the head 30° by raising the head of the bed, and irrigate the ear canal with 10 mL cold water.
    (a) Horizontal nystagmus with the fast component away from the irrigated ear is a normal response, indicating that the brainstem and the cerebral cortex are intact and the patient is not comatose.
    (b) Slow conjugate ipsilateral eye deviation toward the irrigated ear occurs in the comatose patient whose brainstem is functioning.
    (c) No response indicates brainstem dysfunction and most commonly results from structural causes.

C. Essential stabilization and assessment measures

1. Airway management with cervical spine control
2. IV line followed by administration of thiamine 100 mg, glucose 50 g of 50% solution (if hypoglycemic), and naloxone 2–4 mg
3. Oxygen supplementation and pulse oximetry
4. Cardiac monitor
5. Brief history ("what happened"; go through personal effects)
6. Physical and neurologic examinations, including Glasgow Coma Score
7. Complete history (including known allergies)
8. Radiographic clearance of cervical spine (if trauma suspected)
9. Laboratory studies and further radiographic evaluation as indicated

IV. THE ALTERED PATIENT

A. Classic clinical scenario

1. A patient is brought to the emergency department by a concerned relative, friend, or neighbor who has noticed a behavioral change. There is a history of disorientation, forgetfulness, and hallucination.
2. Your task is to determine whether there is organic disease (an underlying medical problem) or a functional (psychiatric) disorder.
   a. Recent onset of a behavioral change with rapid progression suggests an organic cause and can occur in any age group.
   b. The dementias tend to occur in the elderly, and there is a history of gradual onset with slow progression of a behavioral change; however, they can be exacerbated by organic causes.

B. Etiology: organic causes of behavioral change

1. Drug intoxication or withdrawal
2. Metabolic disorders
3. Toxic exposure
4. Cerebrovascular disease
5. Tumors
6. CNS infections (meningitis, encephalitis, cerebral abscess)
7. Anoxia
8. Hypercapnia
9. Uremia
10. Hepatic encephalopathy
11. Vitamin deficiency (B12, thiamine, niacin)
12. Sepsis
13. Neurosyphilis

C. Psychiatric disorders

1. Causes of acute mental confusion
   a. Schizophrenia
   b. Mania
   c. Agitated psychosis
   d. Psychotic depression
   e. Severe adjustment reaction
   f. Posttraumatic/dissociative episodes
2. Dementia: a syndrome of global and progressive deterioration of intellectual function as well as impairment of memory, language, and personality; some of the causes are medically correctable.
   a. Reversible causes (20%)
      (1) Drug toxicity (almost any agent should be suspect)
      (2) Emotional disorders (especially depression)
      (3) Metabolic disorders
      (4) Nutritional deficiencies (especially B_{12})
      (5) Normal-pressure hydrocephalus
      (6) Tumors
      (7) Infection (including non-CNS, eg, pneumonia, urinary tract infection, diverticulitis)
      (8) Complications of arteriosclerosis
      (9) Wilson disease
   b. Irreversible causes (80%)
      (1) Alzheimer disease (50%)
      (2) Multi-infarct dementia (20%)
      (3) Mixed Alzheimer/multi-infarct dementia (10%)
      (4) Rare causes
         (a) Parkinson disease
         (b) Huntington chorea
         (c) Creutzfeldt-Jakob disease
3. Delirium versus dementia
   a. Dementia
      (1) Global cognitive impairment
      (2) Insidious
      (3) Vital signs normal
      (4) Typically elderly
   b. Delirium
      (1) Global cognitive impairment
      (2) Acute onset
      (3) Abnormal vital signs
      (4) Younger individual

D. Clinical presentation: Evaluation requires screening evaluation consisting of history and physical and neurologic examinations, including the Mini–Mental State Evaluation ; never "OMIT" it:

1. Orientation (time and place)
2. Memory (recent and past)
3. Intelligence (thought pattern)
4. Talk (speech pattern)

E. Diagnostic evaluation (directed by physical examination and history)

1. Arterial blood gases
2. CBC and urinalysis
3. Chemistries (glucose, electrolytes, BUN/creatinine)
4. Liver and thyroid function tests
5. Cultures (blood and urine)
6. Toxicology screen (including drug and heavy metals)
7. Chest radiograph
8. CT scan (head)
9. Serum osmolality
10. Serum ammonia
11. Lumbar puncture and evaluation of CSF (if CNS infection suspected)
12. B_{12} levels
13. VDRL or FTA

V. NEUROPATHIES (DISORDERS THAT AFFECT PERIPHERAL NERVES)

A. Etiology (multiple, include toxic, metabolic, and hereditary)

1. Toxins
   a. Heavy metals (lead, arsenic, thallium)
   b. Drugs (eg, amiodarone, dapsone, vincristine)
   c. Industrial solvents
   d. Organophosphates
   e. Diphtheria, tetanus
   f. Ethanol
2. Medical disorders
   a. Guillain-Barré syndrome
   b. Connective tissue disorders
   c. Thiamine deficiency
   d. Hypothyroidism
   e. Tick paralysis
   f. Amyloidosis
   g. Malignancy

h. Diabetes
i. Leprosy
j. Uremia

B. Clinical presentation

1. Most common
   a. Both motor and sensory signs are present.
   b. Weakness and sensory impairment are symmetrical but greater distally than proximally → stocking-glove distribution of sensory symptoms (pain, paresthesia, numbness)
   c. Reflexes are usually absent (Achilles reflex is first affected)
2. Other
   a. A pure motor or sensory loss predominates or
   b. There is proximal (rather than distal) weakness, bulbar symptoms, or asymmetry.

C. Specific toxic neuropathies

1. Diphtheria
   a. Clinical presentation
      (1) An acutely ill patient with fever, tachycardia, and vague history.
      (2) Membranous pharyngitis that bleeds when the membrane is pulled back
      (3) Mononeuritis of the eyes (ptosis, strabismus, accommodation problems)
      (4) Motor involvement in other areas (limbs, palate [usually the first to become paralyzed], urinary, and anal sphincters) may be present; sensory involvement is rare.
2. Tetanus
   a. Clinical presentation
      (1) Symptoms ("4 T's"): trismus (most common), tetany, twitching, and tightness (tight back, arms, and face; a tight facial expression is called "risus sardonicus"); the patient is awake (consciousness is not impaired).
      (2) Signs of sympathetic nervous system hyperactivity (tachycardia, hypertension, hyperpyrexia, sweating) are generally present.
   b. Differential diagnosis
      (1) Dystonic reaction due to phenothiazine therapy
      (2) Poisoning with strychnine
      (3) Local trismus due to odontogenic infection
   c. Treatment
      (1) Human tetanus immune globulin, wound debridement, and antibiotics effective against anaerobes (eg, penicillin G, metronidazole, or a third-generation cephalosporin); clinical disease does not confer immunity, so give the first dose of tetanus toxoid vaccine series in the emergency department.
      (2) Sympathetic hyperactivity may be treated with labetalol.
3. Thallium neuropathy → painful paresthesias, diffuse motor weakness, alopecia (1–2 weeks later)
4. Arsenic neuropathy → polyneuritis (a late finding) with occupational exposure
5. Lead neuropathy → peripheral motor neuropathy (chronic ingestion)
6. Alcohol neuropathy (ethanol, methanol, ethylene glycol) → slowly progressive peripheral neuropathy that is predominantly sensory in a stocking-glove pattern

D. Specific metabolic neuropathies

1. Guillain-Barré syndrome
   a. The most common acute polyneuropathy, this syndrome is an ascending neuropathy preceded by a viral syndrome in 50% of cases.
   b. Clinical presentation: sensory abnormalities (especially paresthesias) → rapid, progressive, symmetrical weakness → paralysis with loss of deep tendon reflexes → respiratory failure
   c. Diagnostic evaluation: CSF reveals cytochemical dissociation (normal cells, → protein)
   d. Miller-Fisher variant: descending symmetrical neuropathy associated with cranial nerve abnormalities
2. Diabetic neuropathy → paresthesias in stocking-glove pattern, poor sensation, muscle weakness
3. Gout neuropathy → extremity neuropathy (particularly of the lumbar plexus)
4. Porphyria neuropathy → polyneuropathy (similar to Gillain-Barré but associated with psychosis and abdominal pain)
5. Tick paralysis → looks like Guillain-Barré syndrome; search for the tick, especially in hairy areas, or in children.

VI. DISORDERS OF THE NEUROMUSCULAR JUNCTION

A. Myasthenia gravis (most common disorder of neuromuscular transmission)

1. Definition: an autoimmune disease that destroys acetylcholine receptors that leads to poor neurotransmission with subsequent proximal muscle weakness
2. Classification
   a. Group 1: localized, nonprogressive disease with perhaps only ptosis or diplopia when fatigued; responds well to anticholinesterase therapy (neostigmine, pyridostigmine); excellent prognosis
   b. Group 2: generalized disease that involves more than one group of striated muscles, both skeletal and cranial in origin, but without sensory involvement; usually amenable to drug therapy; good prognosis
   c. Group 3: acute fulminant disease with severe bulbar manifestations (dysarthria, dysphagia, respiratory failure); poor response to drug therapy and poor prognosis
   d. Group 4: late severe disease that develops at least 2 years after the onset of symptoms in Groups 1 or 2; poor prognosis
   e. Group 5: muscle atrophy that begins ≥6 months after the onset of generalized disease (Group 2); variable prognosis
3. Clinical presentation
   a. Muscle weakness that is exacerbated by activity, sleeplessness, or alcohol intake and is relieved by rest
   b. Ptosis, diplopia, and blurred vision are the most frequent initial symptoms and are the only manifestations of the disease in 20% of cases; the pupil is not involved.
4. Diagnostic evaluation
   a. A double-blind edrophonium test produces transient subjective and objective improvement of symptoms by preventing rapid breakdown of acetylcholine at the myoneural junction.

b. The electromyogram is diagnostic.

c. Serologic testing for antibodies to acetylcholine receptors is useful when positive; however, a negative test does not exclude the disorder.

5. Two forms of acute crisis in myasthenia gravis: myasthenic and cholinergic. Distinction between the two with an edrophonium test may be difficult and hazardous in the extremely weak patient with progressive respiratory insufficiency.

   a. Myasthenic crisis

      (1) Occurs in undiagnosed/untreated patients and those with acute exacerbation of their disease process

      (2) Due to a functional deficiency of acetylcholine

      (3) Can produce severe muscle weakness and respiratory compromise

      (4) Administration of edrophonium 1–2 mg IV (a test dose) followed by 5–8 mg IV → clinical improvement

   b. Cholinergic crisis

      (1) Occurs in patients being treated for myasthenia gravis who receive too much acetylcholinesterase inhibitor

      (2) Due to a functional excess of acetylcholine

      (3) Can produce severe muscle weakness and respiratory compromise.

      (4) Administration of edrophonium → increased muscle weakness. Note whether cardiopulmonary symptoms occur (hypotension, bradycardia, wheezing); if so, atropine 1 mg IV (repeated as needed) should be given but will not improve the signs of neuromuscular blockade.

      (5) Another clue is the presence of muscarinic effects: "SLUDGE" (also suggestive of cholinergic crisis)

      Salivation

      Lacrimation

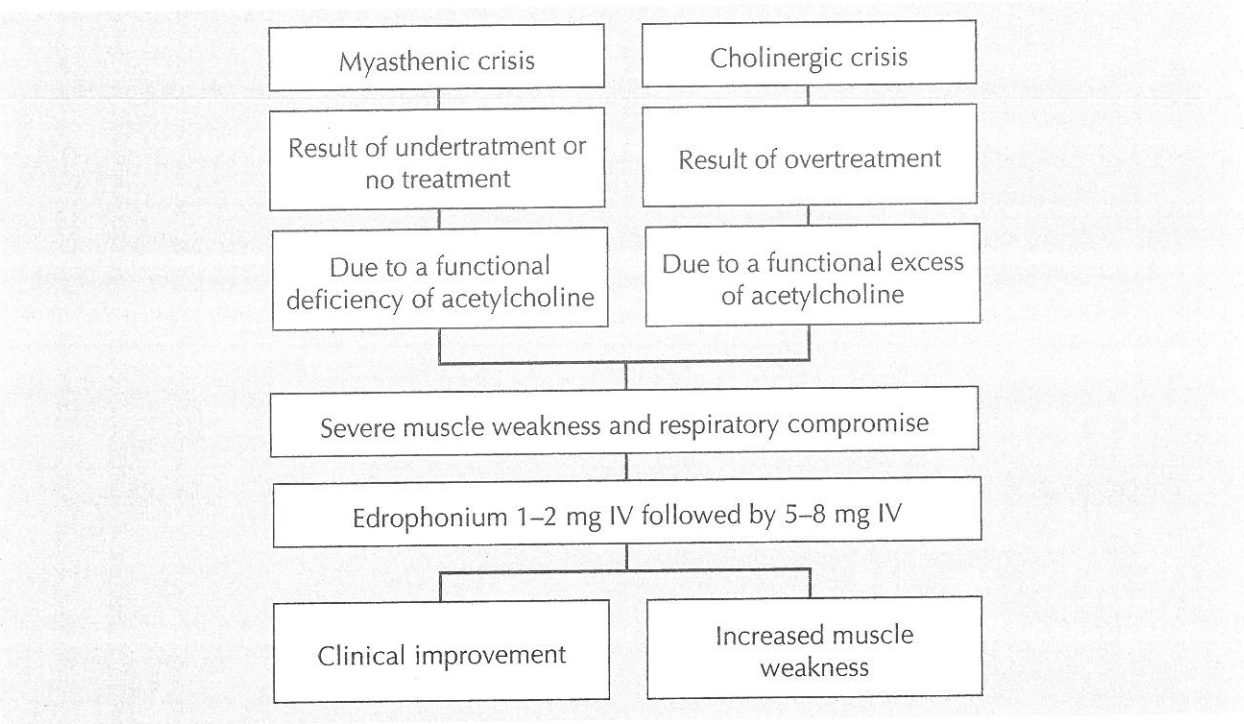
      Urination

      Diaphoresis

      Gastrointestinal cramps

      Emesis

c. Schematic for differentiating the two "crises"



6. Differential diagnosis
   a. Lambert-Eaton syndrome
   b. Botulism
   c. Tick paralysis
   d. Familial periodic paralysis
   e. Sleep paralysis
   f. Amyotrophic lateral sclerosis
   g. Guillain-Barré syndrome
   h. Organophosphate poisoning

B. Lambert-Eaton syndrome

1. A disorder of neuromuscular transmission most often associated with oat cell cancer of the lung → aching, weakness, and fatigability of pelvic girdle and thigh muscles
2. Unlike in myasthenia gravis (which it closely resembles), cranial nerves are generally spared and grip strength increases with repeated stimulation.

C. Botulism (food-borne)

1. Etiology: ingestion of a preformed toxin from *Clostridium botulinum* growing in poorly prepared canned food
2. Pathophysiology: the toxin prevents release of acetylcholine from nerve endings → neurologic symptoms
3. Clinical presentation
   a. Symptoms occur within 24–48 hours: the more severe the poisoning, the earlier the symptoms appear.
   b. The earliest and most common neurologic symptoms (90%) are blurred vision, diplopia (cranial nerves III and VI), and photophobia.

c. Other symptoms that occur within the first 24 hours are dry mouth, dysphagia, and dysarthria.

d. Some symptoms may occur at any time but usually appear early in severe cases → ophthalmoplegia, ptosis, symmetric impairment of cranial nerves and a descending pattern of trunk and extremity weakness; mentation and sensation are normal.

e. Respiratory failure is the usual cause of death and can occur within 6–8 hours of onset of symptoms.

4. Infant botulism ("floppy baby") can occur with the ingestion of raw honey and is associated with acute onset of lethargy, poor feeding, a weak cry, or loss of head control.

VII. MYOPATHIES

A. Differentiating myopathies from neuropathies

Table 19: Characteristics of Myopathies and Neuropathies

Myopathies	Neuropathies
Proximal weakness (getting up from a chair, climbing steps, reaching overhead)	Distal weakness with proximal progression
Sensory symptoms absent	Paresthesias, ↓ sensation (stocking-glove distribution)
Deep tendon reflexes intact	↓ Deep tendon reflexes
Abnormal laboratory tests: ↑ WBC count ↑ erythrocyte sedimentation rate ↑ muscle enzymes	Normal laboratory tests

B. Myopathic syndromes

1. Polymysitis → acute inflammation → muscle pain and weakness → ↑ CPK ± fever
2. Steroid myopathy → muscle pain and weakness associated with chronic steroid therapy
3. Alcoholic myopathy
   a. Acute rhabdomyolysis: binge drinking → distal muscle weakness and tenderness → myoglobinuria and ↑ CPK
   b. Chronic myopathy: steady drinking → proximal muscle weakness and atrophy (no myoglobinuria and CPK is frequently normal)
4. Classic clinical scenario: acute periodic paralysis
   a. You are certain that the diagnosis is "hysteria." The patient is an Asian or Native American male, 7–21 years old who complains of weakness; if you are working the night shift, he may even tell you that the weakness woke him up. Before sending him out, consider this diagnosis and order a serum potassium level (which may be normal, low, or high, because the acute episode is associated with profound hypokalemia due to an intracellular shift of potassium that may have resolved before the patient's arrival in the emergency department).
   b. While waiting for the laboratory result, go back and ask the patient the following: Is there a past history of (or symptoms of) hyperthyroidism? What did he do that day that

was unusual? (A history of extreme physical exertion is characteristic). Has he had previous attacks? What has brought them on, how long did they last, and how did he feel before and after an attack? (The usual story is previous episodes of sudden, severe weakness without associated pain. Cold weather, big meals, trauma and surgery may provoke an attack. The patient feels normal before and after an attack, which usually lasts 1–2 hours. Episodes may be so severe that he may fall while walking or gag while eating.)

c. If the patient's history is suspicious, refer him to a neurologist regardless of the serum potassium level.

5. Hypokalemic myopathy → also causes paralysis, but the etiology and pathogenesis are different from that of periodic paralysis; in this case, the cause is renal tubular acidosis of any etiology (including toluene abuse, Fanconi syndrome) that results in profound potassium depletion.

VIII. ISOLATED PERIPHERAL NEUROPATHIES

A. Trigeminal neuralgia (Tic douloureux)

1. Clinical presentation
   a. Patient complains of excruciating, lancinating pain (like recurrent bursts of an electric shock) that usually follows the distribution of the second or third division of cranial nerve V on the right side of the face (most common).
   b. On physical examination, pain can sometimes be elicited by tapping the trigger zones of the face located along the distribution of the trigeminal nerve (forehead, nose, cheek, chin).
   c. A sensory deficit is unusual (unless the pain is longstanding and there is progressive neuropathy); if present, a coexisting brain tumor should be excluded.
2. Treatment
   a. Long-term medical therapy is with carbamazepine.
   b. Dosage adjustments are required to achieve therapeutic effect, and the patient also needs to be monitored for signs of toxicity; therefore, referral for follow-up is necessary.

B. Bell's palsy

1. Definition
   a. Unilateral facial nerve paralysis (cranial nerve VII) that involves the muscles of the forehead, cheek, and lips
   b. Onset of facial weakness is rapid (within 1–2 days) and is frequently preceded by an upper respiratory infection and 1–2 days of retroauricular pain.
2. It is important to determine if the lesion is located near the origin of cranial nerve VII (brainstem) or somewhere in its peripheral distribution (face only) to determine if a structural lesion or Bell's palsy is present; significant clues of an upper motor neuron lesion are sparing of the muscles of the forehead and slower onset of facial weakness (usually weeks).
3. Differential diagnosis
   a. Other palsies may mimic Bell's palsy.
   b. Ramsay-Hunt syndrome
      (1) Caused by herpes zoster (Bell's palsy may be caused by herpes simplex)

(2) Characterized by typical zoster eruptions on the affected side of the face and neck; if you don't see them on the skin, look for them in the external auditory canal and/or the tympanic membrane.

(3) Cranial nerve VII palsy due to Lyme disease may occur; if it does, it is bilateral (Bell's is unilateral).

4. Bell's palsy is differentiated from a stroke by involvement of the forehead and lower face, the absence of aphasia, hemiparesis, and other focal neurologic deficits.
5. Treatment
   a. High-dose steroids in a short burst followed by gradual tapering with lower doses are effective if given within 48 hours (some say may even be effective if <7 days).
   b. Eye protection (lubricants at night, liquid tears during the day)
   c. Antiviral agents controversial: acyclovir, valacyclovir, famcyclovir should be considered for all patients presenting within 1 week of onset; may improve healing.
   d. ENT referral is essential for all patients.
      (1) Electrostimulatory therapy should be started while the patient is taking steroids.
      (2) Associated findings that constitute an ENT emergency
         (a) Otitis media
         (b) Mastoiditis
         (c) Cholesteatoma
      (3) Other entities should be excluded, because any of them can begin as an isolated lesion of the peripheral seventh nerve.
         (a) Parotid inflammation or tumor
         (b) Middle ear or cranial nerve VIII lesion
         (c) Cerebellopontine angle tumor
         (d) Vascular disease
         (e) Lyme disease
         (f) Ramsey Hunt syndrome (due to herpes zoster)

C. Diabetic mononeuropathies

1. Upper-extremity nerve involvement (commonly seen in the emergency department)
   a. Median
   b. Radial: seen in intoxicated or altered patients who compress their radial nerve while sleeping on it ("Saturday night palsy")
   c. Ulnar: occurs with improper use of crutches, compressing the ulnar nerve
2. Lower-extremity nerve involvement
   a. Femoral
   b. Obturator
   c. Sciatic
3. Examination reveals wasting and tenderness of the involved muscles; associated sensory loss is variable.
4. Oculomotor palsy (cranial nerve III) is the most common cranial neuropathy; the pupil is usually spared (which differentiates this neuropathy from one caused by a tumor or aneurysm).

IX. MYELOPATHIES (DISORDERS OF THE SPINAL CORD)

A. Syringomyelia
   1. Characterized by development of fluid-filled cavity within the spinal cord
   2. Dissociated sensory loss; loss of pain and temperature sensibility with preservation of light touch, vibration, and position; may be impaired in a "capelike" distribution

B. Multiple sclerosis
   1. A demyelinating disorder with myriad potential CNS symptoms, women affected more often than men
   2. Spinal cord involvement results in:
      a. Upper motor neuron signs (weakness, hyperreflexia)
      b. Sensory abnormalities
      c. Bladder/bowel dysfunction
      d. Intranuclear ophthalmoplegia is pathognomonic

C. Transverse myelitis
   1. A postviral or toxic inflammation of the spinal cord (includes radiation effects that may occur during cancer therapy)
   2. Sensation, pain, and temperature sensation are diminished below the level of cord involvement. Vibration and position sense may also be diminished. Patients may have weakness or paralysis of lower extremities.

D. Epidural mass lesion
   1. Clinical presentation
      a. Severe pain (may be the only presenting complaint initially)
      b. Radiating electrical sensations down the spine (Lhermitte sign)
      c. Progressive signs of cord compression
   2. Nontraumatic acute presentations occur in patients with:
      a. Epidural hemorrhage
      b. Acute cervical disc syndromes
      c. Abscess formation (previous IV drug abuse should raise suspicion)
      d. Metastatic tumor

E. Dorsal column disorders
   1. Selective injury to the dorsal columns (classically associated with syphilis and vitamin B12 deficiency).
   2. Results in loss of position sense, vibration, and light touch

X. VOLKMANN ISCHEMIC PARALYSIS

A. Pathophysiology: predisposing condition such as edema, circumferential burn, or tight cast (that results in a compartment syndrome) → ischemia of the nerves and muscles → paralysis with eventual contracture

B. Clinical features of a compartment syndrome (all of which occur late)
   1. Pain out of proportion to the injury
   2. Increased pain with passive stretching of the ischemic muscle compartment
   3. Pallor, paresthesias, and pulselessness (associated findings)

C. Diagnostic evaluation
   1. Diagnosis is confirmed by direct measurement of compartment pressures (which are increased).
   2. Normal compartment pressure is 0–8 mmHg.

D. Treatment: fasciotomy of compartment syndromes will usually prevent this complication.

XI. VERTIGO

A. Definition: the sensation of motion
   1. Subjective vertigo: The patient feels that he or she is moving.
   2. Objective vertigo: The patient feels that the environment is in motion.

B. Types
   1. **Central vertigo**
      a. **Due to lesions of the brainstem or cerebellum**
      b. **Accounts for ~10%–15% of cases and has a more ominous prognosis than peripheral vertigo**
      c. **Clinical presentation**
         (1) **Sudden onset of mild, continuous dysequilibrium that lasts >1 minute (Benign positional vertigo lasts <30 seconds and is brought on by sudden head movements. Any type of vertigo is worsened by changes in head position [and other sensory input] but is not brought on by changes in head position.)**
         (2) **Nausea and vomiting**
            (a) **Mild → pontine lacunar infarct or vertebrobasilar TIA**
            (b) **Severe → acute cerebellar hemorrhage**
         (3) **Hearing loss and tinnitus are rare.**
         (4) **Spontaneous nystagmus is increased by visual fixation and may be bidirectional or vertical.**
         (5) **Positional nystagmus: no latency, does not fatigue or adapt, is multidirectional**
         (6) Associated neurologic abnormalities may be present.
            (a) Severe ataxia, lateralizing dysmetria, or dysdiadochokinesis → cerebellar injury
            (b) **Dysarthria, dysphagia, diplopia, Horner syndrome, motor weakness, scotomata, or blindness → brainstem injury**
      d. **Etiology**
         (1) **Brainstem ischemia or infarction**
         (2) **Cerebellar hemorrhage or infarction**
         (3) **Vertebrobasilar insufficiency**
         (4) **Basilar artery migraine**

(5) Cerebellar or brainstem tumors
(6) Multiple sclerosis
(7) Temporal lobe epilepsy (cortical vertigo)
(8) Posttraumatic or postconcussive syndromes

e. These patients are admitted for further evaluation and treatment.

2. Peripheral vertigo
   a. Due to disorders of the external/middle/inner ear or cranial nerve VIII
   b. Accounts for ~85% of cases and is generally self-limited
   c. Clinical presentation
      (1) Sudden onset of intense, intermittent episodes of dysequilibrium associated with a spinning, whirling sensation
      (2) Associated nausea and vomiting are common.
      (3) Hearing loss and tinnitus are frequently present.
      (4) Spontaneous nystagmus is usually suppressed by visual fixation and is never vertical or bidirectional.
      (5) Positional nystagmus: fatigues and adapts, direction fixed, latency present
      (6) No associated neurologic abnormalities are present.
   d. Etiology
      (1) External ear foreign body or cerumen against the tympanic membrane
      (2) Otitis media or serous otitis
      (3) Ototoxic drugs (eg, trovafloxin, gentamycin, furosemide)
      (4) Vestibular neuronitis
      (5) Benign positional vertigo
      (6) Labyrinthitis or labyrinthine concussion
      (7) Acoustic neuroma
      (8) Meniere disease (endolymphatic hydrops or cochlear hydrops)
         (a) Clinical presentation: Patient is usually 40–60 years old and presents with a history of vertiginous episodes accompanied by tinnitus, sensorineural deafness, and aural pressure. Associated nausea and vomiting are common. Episodes are abrupt in onset and last for minutes to hours.
         (b) Differential diagnosis
            i. Cerebellopontine angle tumor
            ii. Acoustic neuroma
               • Vertigo may not be present because of CNS accommodation for impaired vestibular function.
               ◦ Additional findings: headache, diplopia, decreased corneal sensitivity, facial weakness, positive radiologic findings, increased CSF protein
            iii. Tertiary syphilis
   e. Treatment
      (1) Outpatient with antihistamines or anticholinergics and antiemetics
      (2) Benzodiazepines are effective for symptomatic relief in severe cases.

XII. HEADACHE

A. Pathophysiology

1. Pain-sensitive areas of the cranium are skin, fat, muscles, nerves, the large arteries at the base of the brain, the dural arteries, the falx cerebri, and the great venous sinuses.
2. The major intracranial structures (brain parenchyma, most of the dura, arachnoid, and pia) have no pain fibers.
3. Headache results from tension, traction, inflammation, distention, and dilation of pain-sensitive structures.

B. Types of headache

1. Migraine
   a. Pathophysiology
      (1) Previously thought to be due to vasoconstriction followed by vasodilatation; recent technological advances in biochemistry and pharmacology suggest that this theory is no longer viable.
      (2) Current theories include neurogenic inflammation and abnormalities of the following:
         (a) Serotonergic transmission (5-hydroxytryptamine)
         (b) Trigeminovascular neuronal transmission
         (c) Vascular structures
      (3) Types of auras (represent manifestations of brain dysfunction)
         (a) Visual phenomena (most common), scintillating scotoma, or visual field defects
         (b) Motor disturbances, hemiparesis, ophthalmoplegia, aphasia
         (c) Sensory abnormalities, dysesthesia
         (d) Brainstem disturbances, vertigo, ataxia
   b. Factors that provoke or intensify an attack
      (1) Menstruation (most common bodily change)
      (2) Changes in body rhythm (sleep or food deprivation)
      (3) Physical activity
      (4) Certain foods (chocolate, hard cheese), beverages (red wine or other alcoholic beverages), and chemicals (caffeine, MSG, nitrites)
      (5) Contraceptive estrogens
   c. Clinical presentation
      (1) Migraine without aura (80% of cases)
         (a) The headache is unilateral (initially), pulsating, and usually severe enough to interfere with normal daily activities.
         (b) Associated findings: nausea and vomiting, photophobia, and phonophobia
         (c) A previous history of these attacks (at least five) is required to make the diagnosis.
      (2) Migraine with aura (20% of cases)
         (a) The headache usually follows the aura after a symptom-free period <60 minutes.
         (b) Characteristics of the aura

i. One or more symptoms that are fully reversible
ii. At least one symptom that comes on gradually (>4 minutes) or two symptoms that occur in succession
iii. A single symptom that lasts <60 minutes

(c) A previous history of these attacks (at least two) is required to make the diagnosis.

d. Treatment

(1) Symptomatic therapy

(a) Early abortive therapy

i. NSAIDs (for mild-moderate headache) → ketorolac IM or indomethacin orally or per rectum
ii. Ergotamine orally or per rectum (contraindicated if focal symptoms are present)

(b) Acute phase therapy

i. Prochlorperazine IV
ii. Dihydroergotamine and antiemetic (for moderate to severe headache) → metoclopramide IV or prochlorpromazine IV followed by dihydroergotamine IV or IM (contraindicated if focal symptoms are present)
iii. Sumatriptan SC (contraindicated if focal symptoms are present)
iv. Steroids (for status migrainosus) → hydrocortisone IV followed by prednisone orally in a tapered dosage regimen
v. Opiates → morphine IM repeated in 1–2 hours as needed. Opioids are reserved for those in whom there is no response or standard therapy is contraindicated.

(2) Prophylactic medications (for more than 2 or 3 headaches per month)

(a) ß-blockers (atenolol, propranolol, nadolol, timolol)
(b) Calcium channel blockers may take 3–4 weeks to work.
(c) Serotonin antagonists (cyproheptadine, methysergide)
(d) Cyclic antidepressants (nortriptyline, amitriptyline, doxepin) are effective agents independent of their antidepressant activity in some patients.

2. Cluster headache

a. Classic clinical scenario: The patient is male, at least 25 years old (and frequently a heavy smoker/drinker) who awoke out of sleep with a boring or burning headache on one side of his face; there is either a history of (or signs of) flushing and lacrimation associated with rhinorrhea or nasal stuffiness on the involved side; sometimes there is an associated Horner syndrome (ptosis, miosis, anhydrosis).

b. Treatment

(1) Acute symptomatic therapy

(a) 100% oxygen administered at 7 L/min (effectively treats 75% of patients)
(b) Dihydroergotamine IV or IM and antiemetic IV or sumatriptan SC are effective but should not be given to patients with coronary artery disease.
(c) Instillation of 4% intranasal lidocaine in the ipsilateral nostril is also helpful.

(2) Discharge therapy for patients who are having an exacerbation of cluster headache (if there are no contraindications)

(a) Trial of prophylactic verapamil 80 mg three times per day, to be increased by follow-up physician, and

(b) Tapering course of steroids over 1–2 weeks, which is the typical duration of a cluster headache

3. Subarachnoid hemorrhage (SAH)

a. Clinical presentation

(1) An unusual headache of sudden onset that may have been present for several days; physical examination may be normal, or there may be subtle signs of meningeal inflammation (eg, low-grade fever or mild nuchal rigidity); there may also be slightly abnormal vital signs (↑ blood pressure + ↓ pulse + ↓ respiration = a cerebral event unless proven otherwise)

(2) Sudden onset of a severe "exploding" headache frequently described by the patient as "the worst headache I've ever had" is the classic presentation. Associated nausea and vomiting are common, which may lure the unwary clinician into the "migraine mentality;" a clinical clue is that this headache is unprecedented, ie, unlike any headache the patient has had before, and it is focal.

(3) Neurologic deficits are frequently mild or absent, but nonfocal signs may be present (drowsiness, confusion, personality changes, dizziness, diplopia, or blurred vision).

(4) Sudden, severe headache followed by loss of consciousness → patient presents with hypertension, hyperventilation, and neurologic devastation; subhyaloid hemorrhage may be found on funduscopic examination.

b. An SAH is most commonly due to bleeding from a saccular (berry) aneurysm (~75%) or AV malformation (~10%) but may occur with trauma, vasculitis, and other causes. History often differentiates the two, but occasionally, the hemorrhage precedes a traumatic incident (eg, the patient loses consciousness and then falls, hitting his or her head, or the patient loses consciousness while driving, loses control of the car, and injures himself or herself).

c. Diagnostic evaluation

(1) Brain CT without contrast is the procedure of choice for diagnosing SAH and should be done in any patient with a new onset of a severe or persistent headache. It has a sensitivity of 95% for detecting SAH.

(a) If the CT is negative, a lumbar puncture should be performed because some patients with SAH have a normal CT scan. A yellow supernatant liquid (xanthochromia) that may not be detected for 12 hours, obtained by centrifuging a bloody CSF sample, can help distinguish SAH from a traumatic tap (no xanthochromia). (*ACEP Clinical Policy:* Headaches)

(b) If the diagnosis is still in question, angiography (or repeat lumbar puncture) may be required.

(2) ECG changes (large, broad, or symmetrically inverted T waves, U waves, prolongation of the QRS, prolonged QT interval) may occur in association with SAH and mislead you into pursuing a cardiac diagnosis; dysrhythmias may also result from SAH.

(3) Blood glucose should be determined very early in the evaluation process to exclude hypoglycemia as a cause of an altered mental status.

d. Treatment

(1) Directed toward stabilizing the patient and preventing vasospasm and rebleeding

(2) Obtain immediate neurosurgical consult.

(3) Keep the patient in a quiet room with the lights dimmed.

(4) Elevate the head of the bed 30°.

(5) Start an IV with normal saline or lactated Ringer's (to maintain normovolemia), administer oxygen, place patient on pulse oximeter, and monitor.

(6) If systolic blood pressure is >130 mmHg, consider labetalol.

(7) Provide analgesics and antiemetics (eg, ondansetron) as needed for control of pain, nausea, and vomiting.

(8) Prophylactically treat patient with an anticonvulsant (eg, fosphenytoin) to prevent seizures which can ↑ ICP.

(9) Other therapeutic modalities should be decided in consultation with the neurosurgeon.

(a) Nicardipine may reduce the risk and severity of vasospasm.

(b) Antifibrinolytics (aminocaproic acid) may prevent rebleeding; their use is decreasing because of the associated ischemic complications and the trend toward earlier surgical intervention.

(c) Mannitol or hypertonic saline should be started if patient is showing signs of ↑ ICP.

4. Temporal arteritis (cranial arteritis)

a. Definition: a granulomatous inflammation of one or more branches of the external carotid artery

b. Classic clinical scenario: The patient is usually a woman, often >50 years old, who presents with a severe throbbing, burning headache on one side. There is often a past history of similar headaches that are especially excruciating at night. Associated complaints may include malaise, night sweats, weight loss, blurred vision, and polymyalgia rheumatica. Physical examination often reveals one or more of the following:

(1) A tender, warm, and frequently pulseless temporal artery that can sometimes be rolled between the fingers and skull

(2) Increased burning and decreased throbbing with vessel compression

(3) Decreased visual acuity on the affected side

(4) If some of these findings are absent, be careful not to tag this as "migraine," because failure to diagnose and treat cranial arteritis can result in permanent neurologic loss, especially blindness. Strokes may also occur. The sedimentation rate, by the Westergren method, is characteristically increased (at least >50 mm/hr, often >100 mm/hr); ~25% of patients have only mild increases of the sedimentation rate; temporal artery biopsy (when positive) confirms the diagnosis.

c. Management

(1) Therapy should be started when the diagnosis is suspected; blindness is the most common sequela and may develop rapidly secondary to ischemic papillitis.

(2) Emergent consult with a rheumatologist is necessary to determine disposition and steroid therapy regimen (usually with prednisone in large doses → 60–80 mg/day)

(3) Urgent consult with an ophthalmologist/neurologist or rheumatologist is recommended; if unilateral blindness occurs, loss of vision in the other eye will occur in 1–20 days in 75% of cases.

(4) NSAIDs may be administered for pain relief.

5. **Muscle tension headache: pain is constant and feels like a tight band around the head.**
6. **Space-occupying lesions (brain abscess, tumor): headache is nonthrobbing, prolonged, and constant; onset is gradual, worse in the morning.**
7. **Ocular headache (orbital and/or periorbital pain): may be due to glaucoma, iritis, or corneal abrasion.**
8. **Idiopathic intracranial hypertension (pseudotumor cerebri): affects women more often than men; the patient is usually obese, and headache is usually accompanied by blurred vision and papilledema. CT is negative, MRI excludes dural sinus thrombosis, and lumbar puncture showing ↑ ICP is both diagnostic and therapeutic.**

XIII. MENINGITIS

A. Clinical presentation

1. Acute (pyogenic) meningitis
   a. Rapid onset of symptoms (within 24 hours): fever, headache, photophobia, vomiting, confusion, and stiff neck
   b. Etiology: most commonly bacterial
   c. Begin antibiotic therapy within 30 minutes of presentation.
2. Subacute (lymphocytic) meningitis
   a. Gradual onset of similar symptoms (1–7 days)
   b. Etiology: usually viral
   c. Begin appropriate therapy (usually supportive) within 2 hours of presentation.
3. Chronic meningitis
   a. Insidious onset of symptoms (>1 week)
   b. Etiology: TB, coccidiomycosis, and cryptococcus (most common in HIV patients)
   c. Begin appropriate therapy as soon as the diagnosis is made.
4. Physical examination
   a. Infants (especially neonates) and the elderly typically lack the usual signs and symptoms; in the elderly, the only clue may be an altered level of consciousness.
   b. Look for papilledema, focal neurologic signs (especially ophthalmoplegia), rashes (including purpura and petechiae), and sources of infection.
   c. Brudzinski sign: passive flexion of the neck results in flexion of the hips and knees.
   d. Kernig sign: with the hips flexed, passive extension of the knee (>120°) produces pain.

B. Diagnostic evaluation

1. CT scan of the head
   a. Done first to exclude a mass lesion in patients who have a depressed mental status, seizure, head injury, focal neurologic signs, or papilledema, regardless of whether fever is present.
   b. Antibiotic therapy should not be delayed in patients with suspected bacterial meningitis; antibiotics should be administered before the CT scan.
2. CSF
   a. Characteristics

Table 20: Characteristics of CSF in Different Types of Infections

Test	Normal	Bacterial	Viral	Fungal	TB*
Pressure	<170	>300	<300	300	200
Protein	<50	>200	<200	>200	>200
Glucose	>40	<40	>40	<40	<40
WBCs	<5	>1000	<1000	<500	<1000
Cell type	Monocytes	≥50% PMNs	Monocytes	Monocytes	Monocytes
Gram stain	negative	positive	negative	negative	positive (acid-fast bacilli)

*More common in small children as a manifestation of primary infection but can occur in any age group.

b. CSF antigen and antibody testing

(1) Tests that evaluate the presence of antigen in the CSF (such as latex agglutination studies) are especially useful in the diagnosis of partially treated bacterial meningitis. Demonstration of the CSF cryptococcal antigen is highly sensitive and specific.

(2) Polymerase chain reaction techniques that amplify DNA when performed on CSF, blood, or urine samples are excellent for identification of viral agents; their broader applications in diagnosis of fungal, tuberculous, and bacterial infections are currently being evaluated.

(3) Antibody tests (such as Western blot, ELISA, or complement fixation techniques) are useful in identifying fungal infections (eg, histoplasmosis, coccidiomycosis) or Lyme disease.

(a) A positive CSF VDRL or FTA is 100% specific for a diagnosis of neurosyphilis.

(b) Although expensive and time consuming, ELISA techniques are highly sensitive for diagnosis of viral infections.

C. Etiology and antibiotic therapy

Table 21: Etiology and Therapy of Meningitis in Different Age Groups

Age	Etiology	Antibiotic Therapy*
0–4 weeks old	*E coli*, Group B streptococci, *Listeria monocytogenes*, gram-negative bacilli	Cefotaxime and ampicillin, or gentamicin and ampicillin**
4–12 weeks old	The neonatal pathogens, *Streptococcus pneumoniae, Neisseria meningitides,* and *Haemophilus influenzae*	Cefotaxime and ampicillin, or ceftriaxone and ampicillin**
3 months to 18 years old	*S pneumoniae, N meningitides,* and *H influenzae* (decreasing incidence since *H influenzae* B vaccine)	Ceftriaxone or cefotaxime
18–50 years old	*S pneumoniae, N meningitidis* (decreasing incidence since *H influenzae* B vaccine)	Ceftriaxone
>50 years old and alcoholic patients	*S pneumoniae, L monocytogenes, N meningitidis,* gram-negative bacilli	Ceftriaxone and ampicillin**

*If instituted early, reduces morbidity and sequelae; add vancomycin in any patient >30 days old when *S pneumoniae* resistance is possible.

**If penicillin-allergic, use vancomycin ± rifampin.

D. Steroid therapy

1. The recommendations for steroids are controversial. When given simultaneously with (or before) the first dose of antibiotic, dexamethasone may reduce the incidence of sensorineural hearing loss associated with bacterial meningitis in children; it also decreases the risk of an unfavorable outcome in adults with bacterial meningitis.
2. Indications
   a. Consider in children >6 weeks old with *H influenzae* or *S pneumoniae* meningitis
   b. Adults with proven or suspected pneumococcal meningitis
3. Dosage: 0.15 mg/kg IV

XIV. HYDROCEPHALUS

A. Definition: abnormality in formation, flow, or absorption of CSF that leads to excess accumulation of CSF in the ventricles of the brain

B. Etiology

1. Congenital malformations
2. Mass effect
3. Trauma
4. SAH

C. Clinical presentation: usually a bimodal distribution

1. Children: due to congenital abnormalities; present with vomiting and developmental delay
2. Adults: due to normal-pressure hydrocephalus
   a. Normal-pressure hydrocephalus is defined as a hydrocephalus that has normal opening pressure on lumbar puncture and normal funduscopic examination.
   b. Usually present with ataxia and urinary frequency

D. Treatment

1. Decrease CSF production: acetazolamide or furosemide
2. Repeated lumbar punctures: usually for SAH-induced hydrocephalus, because this may resolve spontaneously after the initial injury heals
3. Surgical management: ventriculoperitoneal or ventriculo-atrial shunt

NERVOUS DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow practice clinical scenarios.

Scenario A

Presentation: A 62-year-old man presents with an acute onset of unilateral headache, followed by right-sided hemiparesis. History includes uncontrolled high blood pressure.

What is the diagnosis?

Scenario B

Presentation: An 18-year-old man complains of peripheral sensory loss that is progressing to ascending paralysis. He had an upper respiratory infection last week.

What is the diagnosis?

Scenario C

Presentation: A 2-week-old female infant presents with irritability, high fever, and vomiting. The fontanelle is full.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: hemorrhagic stroke

Diagnostic evaluation: Nonenhanced (dry) CT of the head is diagnostic and will identify >95% of acute hemorrhagic strokes. MRI ± MRA may be indicated if CT is negative but suspicion is high.

Management: Once the bleed has been identified, acute blood pressure management is indicated. Labetalol and nicardipine IV are first-line treatment options. The goal is to reduce mean arterial pressure by 25% within the first 30 minutes of treatment. Emergent neurosurgical consult is required for evaluation.

Scenario B

Diagnosis: Guillan-Barré syndrome

Diagnostic evaluation: Diagnosis may be difficult in the early stages. The symptoms may be vague and subjective but should not be discounted. CSF analysis should show normal cells but increased protein. Although not diagnostic, inspiratory force (negative inspiratory force) should be measured later in the disease to evaluate respiratory muscle strength.

Management: Patients who have a diagnosis of Guillan-Barré syndrome should be admitted to the hospital for monitoring of respiratory status. Treatments such as intravenous immunoglobulin (IVIG) and plasmaphoresis have limited effect. Most patients recover with supportive care (ie, intubation and enteral feedings) until muscle tone is restored.

Scenario C

Diagnosis: bacterial meningitis

Diagnostic evaluation: Neonates may present with fever and be inconsolable. In older patients, altered mental status may be the only symptom. A Gram stain of CSF obtained by lumbar puncture is the "gold standard." Performing a CT scan before lumbar puncture is not necessary if the patient has normal neurologic and funduscopic examinations. CT scan may identify alternative reasons for this presentation, such as intracranial abscess or SAH.

Management: The patient should be treated with empiric antibiotics based on the most likely causative organism. Double coverage with vancomycin is warranted if there is suspicion of methicillin-resistant *Staphylococcus aureus* (MRSA). If clinical suspicion is high, treatment should begin before verification of diagnosis. Pretreating the patient with steroids is controversial, but it may help alleviate some of the long-term morbidity associated with the disease.

Table 22: Etiology and Therapy of Meningitis in Different Age Groups

Age	Etiology	Antibiotic Therapy*
0–4 weeks old	*E coli*, Group B streptococci, *Listeria monocytogenes*, gram-negative bacilli	Cefotaxime and ampicillin, or gentamicin and ampicillin**
4–12 weeks old	The neonatal pathogens, *Streptococcus pneumoniae*, *Neisseria meningitides*, and *Haemophilus influenzae*	Cefotaxime and ampicillin, or ceftriaxone and ampicillin**
3 months to 18 years old	*S pneumoniae*, *N meningitides*, and *H influenzae* (decreasing incidence since *H influenzae* B vaccine)	Ceftriaxone or cefotaxime
18–50 years old	*S pneumoniae*, *N meningitidis* (decreasing incidence since *H influenzae* B vaccine)	Ceftriaxone
>50 years old and alcoholic patients	*S pneumoniae*, *L monocytogenes*, *N meningitidis*, gram-negative bacilli	Ceftriaxone and ampicillin**

*If instituted early, reduces morbidity and sequelae; add vancomycin in any patient >30 days old when *S pneumoniae* resistance is possible.

**If penicillin-allergic, use vancomycin ± rifampin.

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Note: This section includes many antibiotics and dosing regimens. You are not expected to memorize them, but you should be familiar with them so that you will recognize specific antibiotics and corresponding dosages when they appear on the examination.

GYNECOLOGIC AND OBSTETRIC DISORDERS: SELF-ASSESSMENT QUESTIONS

1. Which of the following statements regarding syphilis, chancroid, lymphogranuloma venereum, and genital herpes simplex is most accurate?

   (a) Treatment of these sexually transmitted infections should be withheld until laboratory confirmation has been obtained.
   (b) Doxycycline is the recommended drug of choice for each of these infections.
   (c) Patients with these infections commonly complain of a vaginal or penile discharge.
   (d) These infections are characterized by genital ulcers and/or inguinal lymphadenopathy.

2. A female patient presents with the complaint of an itchy, malodorous vaginal discharge that is yellow-gray in color. On examination, her vaginal mucosa has a stippled appearance, and the vaginal pH is 6.0. Wet mount reveals motile pear-shaped organisms with flagella at one end. The patient tells you she is 5 weeks pregnant. How would you treat her for this condition?

   (a) Prescribe metronidazole 2 g orally in one dose.
   (b) Recommend vaginal douching every night with yogurt × 7 days.
   (c) Prescribe clotrimazole vaginal suppositories bid × 7 days.
   (d) Recommend vinegar douches every night × 7 days.

3. A pregnant patient presents in her first trimester with a foul-smelling vaginal discharge. Examination reveals diffuse vaginal erythema and foamy grayish yellow-green discharge. Wet mount reveals many PMNs and flagellated, motile, tear-drop–shaped organisms. The most appropriate therapy is:

   (a) Metronidazole
   (b) Doxycycline
   (c) Clotrimazole cream or suppositories
   (d) Clindamycin

4. Which of the following is not considered a risk factor for pelvic inflammatory disease?

   (a) Use of an intrauterine device
   (b) Use of birth control pills
   (c) Multiple sexual partners
   (d) Adolescence

5. The most common cause of pelvic inflammatory disease in college students is:

   (a) *Chlamydia*
   (b) *Neisseria gonorrhoeae*
   (c) Anaerobic bacteria
   (d) Genital tract mycoplasmas

6. All of the following are considered safe for administration during pregnancy except:

   (a) Tetanus toxoid
   (b) Penicillins/cephalosporins
   (c) Erythromycin (except for estolate)
   (d) Measles vaccine

7. All of the following statements regarding vulvovaginal candidiasis are accurate except:

   (a) *Candida albicans* is part of the normal vaginal flora in up to 20% of women.
   (b) Causes of colonization or overgrowth include diabetes mellitus, pregnancy, menstruation, and birth control pills.
   (c) Diagnosis is with a potassium hydroxide prep that reveals "clue cells."
   (d) Treatment of partners is generally unnecessary, because this is not a sexually transmitted infection.

8. A young, sexually active woman presents with a history of right lower quadrant pain for the past several hours. Her last menstrual period was 6 weeks ago, but she says that is not unusual for her. Her vital signs are stable, but she looks visibly uncomfortable. On abdominal examination, you note tenderness confined to the right lower quadrant, and pelvic examination reveals right adnexal tenderness and fullness. Which of the following possible diagnoses is least likely?

   (a) Ectopic pregnancy
   (b) Pelvic inflammatory disease
   (c) Hemorrhagic corpus luteum cyst
   (d) Adnexal torsion

9. In the patient discussed in question #8, which of the following laboratory studies would be least likely to help you in narrowing the differential and establishing a definitive diagnosis?

   (a) CBC
   (b) Culdocentesis
   (c) ß-human chorionic gonadotropin
   (d) Pelvic ultrasound

10. The incidence of transmission of rubella from an infected mother to her fetus is greatest when maternal infection occurs:

    (a) In the first month of pregnancy
    (b) In the second month of pregnancy
    (c) In the third month of pregnancy
    (d) Is the same in the first 3 months of pregnancy

11. The drugs considered to be safest for administration during pregnancy are those classified by the FDA as:

    (a) Category A drugs
    (b) Category C drugs
    (c) Category D drugs
    (d) Category X drugs

12. All of the following statements regarding the diagnosis of intrahepatic cholestasis of pregnancy are accurate except:

    (a) Patients usually present in their third trimester with pruritus and mild jaundice.
    (b) Associated laboratory findings include a 7- to 10-fold increase in the alkaline phosphatase level and markedly increased serum transaminases.
    (c) Symptoms are treated with cholestyramine.
    (d) It is associated with an increased risk of premature delivery and fetal compromise.

13. Which of the following is not considered to be a risk factor for abruptio placenta?

    (a) Hypertension
    (b) Abdominal trauma
    (c) Primiparity
    (d) Smoking

14. A patient is brought in by ambulance for evaluation. The paramedics state that she had been out shopping with a friend and had a grand mal seizure. A fingerstick done in the field showed a glucose level of 120 mg/dL. There is no known history of a prior seizure disorder. On evaluation, she looks to be about 17 years old and at least 8 months pregnant. She is lethargic and hyperreflexic (+ ¾) and has a blood pressure of 170/100 mmHg. The most appropriate initial medication is:

    (a) Hydralazine
    (b) Diazepam
    (c) Furosemide
    (d) Magnesium sulfate

15. Signs of magnesium toxicity include all of the following except:

    (a) Loss of deep tendon reflexes
    (b) Respiratory depression
    (c) Flushing
    (d) Bradydysrhythmias



16. Appropriate initial evaluation of a woman with vaginal bleeding in the third trimester includes all of the following except:

    (a) Evaluation of the fetal heart tones
    (b) Pelvic examination
    (c) CBC, type and Rh, disseminated intravascular coagulation profile
    (d) Ultrasound

17. A woman who is 34 weeks pregnant presents with right upper quadrant pain of several hours duration. On examination, she has a temperature of 100.4°F (38°C) and diffuse right upper quadrant tenderness. CBC reveals a WBC count of 17,000/mm^3. Alkaline phosphatase and liver function tests are normal, and urinalysis reveals pyuria without bacteria. The most likely diagnosis is:

    (a) Pyelonephritis
    (b) Salpingitis
    (c) Appendicitis
    (d) Cholecystitis

18. A woman who is 4 days postpartum presents with vaginal bleeding. Examination reveals a temperature of 101°F (38.3°C), a purulent discharge, and uterine tenderness. The most likely cause of the postpartum bleeding is:

    (a) Retained placental tissue
    (b) Endometritis
    (c) Uterine atony
    (d) Genital tract trauma sustained during delivery

19. All of the following statements regarding the Kleihauer-Betke test are accurate except:

    (a) It can be used to detect and quantify fetomaternal hemorrhage.
    (b) It is useful in determining the amount of Rh immune globulin administered to an Rh-negative woman who has sustained fetomaternal hemorrhage.
    (c) Its use is generally limited to women who are at least 12 weeks pregnant.
    (d) A negative test means that no fetomaternal hemorrhage has occurred.

ANSWERS

1. d	6. d	11. a	16. b
2. a	7. c	12. b	17. c
3. a	8. b	13. c	18. b
4. b	9. a	14. d	19. d
5. a	10. a	15. c	

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

SEXUALLY TRANSMITTED INFECTIONS

I. GONORRHEA

A. Clinical presentation (may be asymptomatic)

1. In men, the usual complaint is a purulent urethral discharge that is frequently associated with dysuria. Rectal gonorrhea (venereal proctitis) should be suspected in the presence of a rectal discharge in men who have sex with men.
2. In women, the disease may be misdiagnosed in the early stages as nonspecific vaginitis, cervicitis, or urinary tract infection because the presenting complaint is frequently dysuria, a vaginal discharge, or both. Abdominal pain does not occur until the infection has spread to the uterus and fallopian tubes (pelvic inflammatory disease).
3. Pharyngeal or anal infection may occur in either sex. Although generally asymptomatic, patients with anal infection may complain of discharge, rectal pain, tenesmus, and/or constipation. Patients with pharyngeal infection are rarely symptomatic.
4. Gonococcal conjunctivitis is usually the result of inoculation of the eye by a contaminated finger. A copious purulent discharge, marked conjunctival injection, and unilateral involvement are characteristic.
5. Hematogenous dissemination also occurs in both sexes and may produce any of the follow complications:
   a. Joint disease
      (1) Most common cause of septic arthritis in patients <50 years old, especially teenagers and young adults
      (2) Diagnosis should be considered in any young patient with joint pain. A classic (but not necessarily common) presentation is a fever, rash, and multiple joint pain that includes a tenosynovitis.
   b. Endocarditis
   c. Sepsis
   d. Meningitis
   e. A rash may be associated with any of these complications but is most frequently seen if fever is present. The skin lesions of gonococcemia are typically erythematous macules or pustules with a necrotic or purpuric center. Petechiae are also seen.

B. Diagnostic evaluation

1. Gram-negative intracellular diplococci seen on Gram-stain smears from skin lesions, urethral discharge in men, or cervical discharge in women; this provides a tentative diagnosis of gonorrhea.
2. *Neisseria gonorrhoeae* should be identified on culture (considered the "gold standard" method of diagnosis) of suspected lesions or discharges. It can also be diagnosed by nucleic acid amplification tests (polymerase chain reaction/ligase chain reaction of DNA).
3. Culture is required for diagnosing organisms from the oropharynx, synovial fluid, anorectal area, cerebrospinal fluid, and in medicolegal cases.
4. A positive smear or culture can be difficult to obtain. Repeated cultures should be done if clinical suspicion remains.

5. Nucleic acid amplification tests have a sensitivity of >90% (versus 60%–80% for culture) and a specificity of >99%.
6. In symptomatic patients, nucleic acid amplification tests of endocervical swabs from women or urethral swabs from men are good alternatives to gonococcal cultures.

C. Management

1. Start as soon as the diagnosis is suspected clinically or there is a history of exposure. Do not wait for culture results, because this may result in the development of more advanced disease and continued transmission of the infection.
2. Because of the prevalence of concurrent chlamydial infection (up to 45% of patients with gonorrhea have a coexisting chlamydial infection), all patients should receive simultaneous antibiotic coverage for both *N gonorrhoeae* and *Chlamydia*.
3. Given the increasing incidence of penicillinase-producing *N gonorrhoeae* and tetracycline-resistant *N gonorrhoeae*, ceftriaxone is currently the drug of choice for treatment of gonorrhea. Of note, the currently recommended dosage of ceftriaxone (250 mg) is higher than the previously recommended dosage (125 mg).
4. Drug regimen of choice for uncomplicated infections (no hematogenous dissemination)
   a. Ceftriaxone 250 mg IM (may be used in pregnant patients) plus
   b. Doxycycline 100 mg orally bid × 7 days or azithromycin I g orally
      (1) Effective in treating concurrent chlamydial infection as well as incubating syphilis
         (a) If a gonorrhea culture is negative, *Chlamydia* trachomatis is the most likely offending organism (because it is the most common sexually transmitted infection).
         (b) Untreated chlamydial pelvic inflammatory disease can lead to tubal injury with subsequent infertility problems.
         (c) Epididymitis in men <35 years old without a urethral discharge is most commonly a chlamydial infection and is considered to be the most common sexually transmitted infection in men.
      (2) Doxycycline is contraindicated in pregnancy and should not be used in children <8 years old. In these patients, erythromycin base 500 mg orally qid × 7 days may be substituted for doxycycline.
   c. Alternative drug therapy for uncomplicated infections for patients who cannot take ceftriaxone
      (1) Cefixime 400 mg orally (single dose) plus doxycycline 100 mg orally bid × 7 days *or*
      (2) Azithromycin 2 g orally; limit to patients with documented allergy due to increasing resistance patterns.
   d. Alternative drug therapy for uncomplicated infections in patients who cannot take cephalosporins: spectinomycin 2 g IM (may be used in pregnant patients)
5. Patients with gonococcal conjunctivitis should be hospitalized and also receive timely ophthalmologic consultation.
6. Patients with disseminated gonorrhea should be hospitalized for initial therapy.
7. Advise treatment of sexual contacts (even if signs and symptoms of active disease are absent).
8. Recommend abstinence until sexual partners have been evaluated and treated, and antibiotic therapy has been completed.
9. Obtain a serologic test for syphilis (RPR or VDRL) in all patients evaluated and treated for gonorrhea.
10. Recommend a follow-up culture 1 week after the completion of therapy for patients who have symptoms that persist after treatment, or treat with alternative regimens.

II. CHLAMYDIA

A. Etiology

1. Caused by *Chlamydia trachomatis*, an obligate intracellular parasite that causes a range of disease from cervicitis to pelvic inflammatory disease
2. It is estimated that 1 in 15 sexually active teenagers has *Chlamydia*.

B. Clinical presentation

1. "Typical" clinical presentation: patients may be asymptomatic or have vaginal irritation and discharge, or fulminant pelvic inflammatory disease. Presentation is similar to that of gonorrhea.
2. Ocular *Chlamydia* may occur and cause blindness; transmission is typically from mother to fetus upon passage through birth canal.
3. May also be found in rectum and oropharynx

C. Diagnostic evaluation

1. Pelvic examination with cultures and wet prep should be completed for women; endocervical swab should be taken.
2. Men may be diagnosed through either a urethral swab or urine specimen.
3. Nucleic acid amplification test is more sensitive than culture.
4. Chlamydial cervicitis should be suspected if Gram stain reveals PMNs but no organisms.

D. Management

1. Drug regimen
   a. Azithromycin 1 g orally once or
   b. Doxycycline 100 mg orally twice a day × 7 days
2. Alternative therapy
   a. Erythromycin base 500 mg orally four times a day × 7 days
   b. Erythromycin ethylsuccinate 800 mg orally four times a day × 7 days
   c. Levofloxacin 500 mg orally once daily × 7 days
   d. Ofloxacin 300 mg orally twice a day × 7 days
3. Start as soon as the diagnosis is suspected clinically or there is a history of exposure. Do not wait for culture results, because this may result in the development of more advanced disease and continued transmission of the infection.
4. If untreated, may lead to infertility and ectopic pregnancy.
5. Sexual partners need to be treated.

III. SYPHILIS

A. Etiology

1. A common sexually transmitted infection (especially in men who have sex with other men) caused by a spirochete (*Treponema pallidum*) found in infected skin lesions, mucous membranes, saliva, semen, and blood.

2. Transmission occurs primarily by direct contact with an infected lesion (usually genital). Syphilis may also be acquired congenitally or by a blood transfusion (if the donor was in a very early stage of the disease).

B. Clinical phases and diagnostic evaluation

1. Primary syphilis
   a. A small papule develops at the site of inoculation (usually genital) and becomes a painless, indurated ulcer—the classic chancre. Chancres develop after an incubation period of 10–90 days, are present for 3–6 weeks, and resolve spontaneously.
      (1) Scrapings obtained from the chancre and examined under darkfield microscopy reveal moving corkscrew-like treponemes.
      (2) Serologic tests (VDRL or RPR) are often negative.
   b. Inguinal lymphadenopathy (buboes) may be present. Nodes are enlarged, nontender, firm, and rubbery; they develop ~4 weeks after the initial exposure.
      (1) Material obtained from the nodes is dark-field positive.
      (2) Serologic tests are usually positive.
2. Secondary syphilis
   a. Characterized by constitutional signs/symptoms and a rash
   b. Rash and lymphadenopathy are the most common symptoms.
   c. The rash of secondary syphilis emerges 4–8 weeks after the initial appearance of the chancre and lasts for several months. It may be difficult to distinguish from the rash of Rocky Mountain spotted fever, because both are seen on the palms and soles. Definitive differentiation is with serologic testing (VDRL, RPR, or FTA-ABS).
      (1) Erythematous (red) macules, papules, or plaques (condylomata lata) suggest syphilis. The classic rash is maculopapular, nonpruritic, and symmetric, and the palms and soles are commonly involved.
      (2) A fine pink (not red) rash interspersed with macules suggests Rocky Mountain spotted fever.
3. Tertiary syphilis
   a. Onset occurs after a latent (dormant) period of several years
   b. Characterized by development of cardiovascular and/or neurologic disorders such as thoracic aneurysm, dementia, and tabes dorsalis
   c. Serology tests are positive.
   d. Neurosyphilis should be considered in the differential diagnosis of HIV-positive patients with neurologic disease.

C. Management

1. Exposed patients should be evaluated clinically and serologically, and then treated on a presumptive basis.
2. All patients who have syphilis should be tested for HIV infection.
3. Sexual partners should be notified and treated.
4. Penicillin G is the treatment of choice for syphilis, regardless of stage; doxycycline is the alternative choice for the penicillin-allergic patient.
5. Primary, secondary, and early latent (<1 year duration) syphilis
   a. Benzathine penicillin G 2.4 million units IM once *or*
   b. Doxycycline 100 mg orally bid × 14 days (nonpregnant patients) *or*

c. Tetracycline 500 mg orally qid × 14 days (nonpregnant patients) *or*
d. Ceftriaxone 1 g/day IM or IV × 8–14 days
e. Azithromycin 2 g orally, effective for treating early syphilis only
f. Pregnant patients who are allergic to penicillin should be desensitized and treated with penicillin.

6. Tertiary syphilis
   a. Cardiovascular syphilis and/or late latent syphilis (>1 year duration)
      (1) Benzathine penicillin G 2.4 million units IM weekly for 3 weeks *or*
      (2) Doxycycline 100 mg orally bid × 4 weeks (nonpregnant patients) *or*
      (3) Tetracycline 500 mg orally qid × 4 weeks (nonpregnant patients)
      (4) Pregnant patients who are allergic to penicillin should be desensitized and treated with penicillin.
   b. Neurosyphilis
      (1) Aqueous crystalline penicillin G 3–4 million units IV every 4 hours × 10–14 days or
      (2) If compliance assured → procaine penicillin G 2.4 million units/day IM plus probenecid 500 mg orally qid, both × 10–14 days. Penicillin-allergic patients require desensitization followed by one of the above regimens or ceftriaxone 2 g/day IM or IV × 10–14 days (for patients without a prior anaphylactic reaction to penicillin).
7. Follow-up serologic testing (VDRL or RPR) should be recommended, because treatment failure may occur with any drug regimen.
8. Jarisch-Herxheimer reaction
   a. A self-limited, acute febrile reaction accompanied by headache, myalgias, and rash intensification
   b. Usually occurs 1–2 hours after antibiotic therapy is started (certainly within the first 24 hours) and is treated with antipyretics and bed rest

IV. CHANCROID

A. Etiology

1. Although chancroid is the least reported sexually transmitted infection, the incidence is rising.
2. The infecting organism, *Haemophilus ducreyi*, a small, gram-negative bacterium, is acquired through sexual contact from discharges of ulcerations or suppurating lymphatic tissue; the incubation period is 2–10 days.

B. Clinical presentation

1. The initial sign is a small pustule or papule that quickly breaks down into one or more painful chancres.
2. "Pain triad"
   a. Painful necrotic chancre (dirty ulcer, ragged)
   b. Painful urination on contact with the chancre
   c. Painful inguinal lymphadenopathy that is suppurative and frequently associated with extensive tissue destruction; bubo formation (50% of cases) with spontaneous rupture is common.

C. Diagnostic evaluation

1. If the laboratory has experience with this organism, obtain an aspirate from the inguinal bubo and send it for Gram stain and culture.
   a. Gram stain reveals short, gram-negative bacilli in a linear or parallel formation ("school of fish").
   b. A positive culture confirms the diagnosis, but a negative culture does not exclude the diagnosis.
2. If the laboratory does not have the capability to isolate this organism, diagnosis should be based on clinical presentation.
3. HIV testing should be done if chancroid is suspected; there is a high rate of co-infection.
4. Herpes and syphilis must also be excluded by testing.

D. Management

1. Azithromycin l g orally in a single dose *or*
2. Ceftriaxone 250 mg IM in a single dose *or*
3. Ciprofloxacin 500 mg orally bid × 3 days (quinolones are contraindicated in pregnancy and in patients <17 years old) *or*
4. Erythromycin base 500 mg orally tid × 7 days or until lesions are healed
5. All four regimens are effective for the treatment of chancroid in HIV-infected patients.
6. Advise treatment of sexual contacts, even if they are asymptomatic.
7. Recommend abstinence until sexual partners have been treated and antibiotic therapy has been completed.

V. LYMPHOGRANULOMA VENEREUM

A. Etiology and epidemiology

1. Uncommon sexually transmitted infection caused by *Chlamydia trachomatis*
2. Usually seen in men 20–40 years old

B. Clinical presentation

1. A small, shallow, painless vesicle or ulcer appears 3–21 days after exposure. It usually goes unnoticed by the patient.
2. Localized, tender inguinal (and/or femoral) lymphadenopathy that is usually unilateral develops 2–24 weeks after the exposure.
3. When the nodes enlarge, coalesce, and ulcerate, constitutional symptoms may develop.
4. The "groove sign" may appear and is characterized by the proliferation of inguinal lymphadenopathy above and below the inguinal ligament.

C. Diagnostic evaluation

1. The lymphogranuloma venereum complement fixation test (which has replaced the Frei skin test) is diagnostic but not available in many hospital laboratories.
2. Culture is also diagnostic, but many laboratories do not have this capability.
3. The diagnosis is usually made by identification of WBCs with intracellular inclusion bodies from aspirates of infected tissue.

D. Management

1. Treatment should be started as soon as the diagnosis is suspected, without waiting for laboratory confirmation.
2. Doxycycline 100 mg orally bid × 3 weeks is the treatment of choice but should be avoided in pregnancy, while nursing, and in children <8 years old. Pregnant women should be treated with erythromycin (not the estolate formulation) 500 mg orally qid × 3 weeks.
3. Treatment of sexual partners should be recommended.
4. HIV testing is recommended in patients with suspected lymphogranuloma venereum.

VI. TRICHOMONIASIS

A. Definition and etiology

1. A common, although minor, sexually transmitted infection caused by the flagellated protozoan *Trichomonas vaginalis* that is transmitted by contact (usually genital) with infected secretions of the genital and urinary tracts
2. The incubation period is usually a week, with symptoms occurring in women but not men.

B. Clinical presentation

1. Patients complain of a malodorous, itchy, vaginal discharge that is typically frothy or foamy.
2. On examination, the discharge may be yellow or have a gray-green appearance; the vaginal mucosa and cervix may have a stippled or punctate "strawberry" appearance. The vaginal pH is usually 5.5.

C. Diagnostic evaluation

1. Wet mount and direct visualization of the motile trichomonads, which are pear-shaped and have several flagella at one end
2. Culture is more sensitive.

D. Management

1. Metronidazole 2 g (single dose) or 500 mg bid × 7 days or tinidazole 2 g orally (single dose) are equally effective.
   a. Use metronidazole for the 7-day regimen if single-dose therapy fails.
   b. For repeated failures, use 2 g/day × 3–5 days.
2. Sexual partners should be treated.
3. Advise patients to avoid ingestion of alcoholic beverages during treatment with metronidazole and for at least 24 hours thereafter, because ingestion may precipitate a disulfiram ("Antabuse") reaction (flushing, headache, nausea, vomiting, palpitations, and abdominal discomfort).

VII. HERPES SIMPLEX

A. Etiology

1. A common sexually transmitted infection caused most often by type 2 (80%) herpes simplex virus and occasionally by type 1 (20%).

2. Transmission is generally by sexual contact with an infected individual; it may occur in the absence of visible lesions if viral shedding is present.
3. Incubation period is 8–16 days.

B. Clinical presentation

1. Recurring, painful genital lesions appear as clusters of vesicles on an erythematous base and subsequently denude forming ulcers.
2. The lesions may be found at any site, but the most commonly affected areas are:
   a. The cervix and vulva in women (swelling of the vulva may be so severe that the patient may not be able to void urine)
   b. The glans and prepuce in men
3. Attacks are often heralded by a prodrome of pain, hyperesthesia, burning, or paraesthesia at the skin site.
4. Headache, fever, arthralgias, and regional adenopathy may also be present, particularly with the first clinical episode.
5. Newborns who acquire the virus at birth may develop devastating complications.

C. Diagnostic evaluation

1. The clinical findings are usually sufficient evidence of the diagnosis.
2. If there is some question, the diagnosis can be confirmed by one of the following:
   a. Direct immunofluorescence testing
   b. Viral culture is the gold standard. Test takes 3–10 days to complete. False-negative rate is 5%–20%.
   c. New type-specific viral serologic testing is the recommended test for newly presenting patients.
3. The Tzanck smear used in the past for diagnosis of herpes simplex virus is no longer recommended because of its lack of sensitivity.

D. Management

1. Acyclovir (oral or IV) remains the antiviral agent of choice.
   a. Accelerates healing
   b. Shortens the duration of viral shedding
   c. Provides partial control of symptoms
   d. Topical acyclovir is not recommended.
   e. Famciclovir and valacyclovir are acceptable alternatives.
   f. Foscarnet is often effective for treatment of acyclovir-resistant genital herpes.
   g. Neither acyclovir nor any other treatment regimen has succeeded in preventing the recurrence of lesions.
2. Indications for treatment with oral acyclovir
   a. Patients with primary lesions
   b. Patients with associated herpes cervicitis, urethritis, proctitis, stomatitis, or pharyngitis
   c. Patients with recurrent genital herpes
   d. Episodic infections in HIV-positive patients
3. Indications for hospital admission and treatment with IV acyclovir
   a. Patients with severe genital infections

b. Immunocompromised patients with severe infections/disseminated disease
c. Patients who cannot tolerate or do not respond to oral medication
d. Neonatal herpes

4. Dosing guidelines
   a. First episode
      (1) Acyclovir 400 mg orally tid × 7–10 days or
      (2) Acyclovir 200 mg orally 5 times per day × 7–10 days or
      (3) Famciclovir 250 mg orally tid × 7–10 days or
      (4) Valacyclovir 1 g orally bid × 7–10 days
   b. Recurrent episodes
      (1) Acyclovir 400 mg orally tid × 5 days or
      (2) Acyclovir 800 mg orally bid × 5 days or
      (3) Acyclovir 800 mg orally tid × 2 days or
      (4) Famciclovir 125 mg orally bid × 5 days or
      (5) Famciclovir 1,000 mg orally × bid × 1 day or
      (6) Valacyclovir 1 g/day orally × 5 days or
      (7) Valacyclovir 500 mg orally bid × 3 days
5. Prescribe analgesics as needed.
6. If an associated cellulitis is present, prescribe antistaphylococcal antibiotics.
7. Advise patients to avoid sexual contact when lesions are present.

E. Complications

1. Herpes simplex virus has been epidemiologically associated with both cervical cancer and cancer of the vulva.
2. Genital herpes has been associated with an increased risk of acquiring HIV infections.
3. Cesarean section is indicated in term-pregnant patients with active genital herpes because of the high neonatal mortality rate (50%–80%) and morbidity associated with contraction of neonatal herpes during vaginal delivery.

VIII. HUMAN PAPILLOMA VIRUS

A. Etiology

1. Herpes simplex virus has >40 serotypes.
2. Linked to the development of genital warts as well as cervical cancer

B. Clinical presentation

1. Frequently an asymptomatic infection, although clinical presentation depends on the serotype and location of infection.
2. The virus may infect the oropharynx, genital, or anal tissue.
3. Genital warts are usually flat papules or pedunculated and skin colored.
4. In children, papillomas can form in the respiratory tract.

C. Diagnostic evaluation

1. Diagnostic evaluation should include genital examination in the emergency department for genital warts. Emergency concerns are those of large, obstructing lesions.
2. Can cause abnormal findings on pap smears, cervical cancer

D. Management and prevention

1. External lesions: home treatment may include podofilox 0.5% solution or gel or imiquimod 5% cream. Alternatively, the patient may be referred to a specialist for cryotherapy, trichloracidic acid, or surgical removal.
2. Appropriate cancer screening and follow-up is recommended.
3. Vaccination is recommended for children 11–12 years old to prevent development of cancer. (Vaccination is not typically done in the emergency department.)

GYNECOLOGIC DISORDERS

I. VULVOVAGINITIS (INFLAMMATION OF VAGINA AND EXTERNAL GENITALIA)

A. *Trichomonas* vaginitis (a sexually transmitted infection)

1. Etiology: *Trichomonas vaginalis*, a flagellated protozoan
2. Clinical presentation
   a. Patients present with vulvovaginal itching and a foul-smelling discharge.
   b. Associated dysuria and lower abdominal pain may also be present. May be responsible for 20% of nongonococcal urethritis in men.
   c. Symptoms usually develop after an incubation period of 4–28 days.
   d. Profuse, occasionally foamy, yellow-green discharge associated with diffuse vaginal erythema and a "strawberry cervix and vagina" (punctate hemorrhages)
3. Diagnostic evaluation
   a. Wet mount demonstrates a flagellated, motile, tear-drop–shaped organism and many PMNs. Sensitivity 60%–70%.
   b. Polymerase chain reaction testing is available for point-of-care testing and has higher sensitivity and specificity than wet mount.
   c. Vaginal pH ≥5.5
4. Management (partner must also be treated)
   a. Recommended regimen (includes pregnant patients): metronidazole 2 g orally in one dose or tinidazole 2 g orally in one dose
   b. Alternative regimen: metronidazole 500 mg orally bid × 7 days
   c. Metronidazole can produce a disulfiram ("Antabuse") reaction if taken in conjunction with alcohol. Therefore, patients must be advised to avoid ingesting alcohol while taking metronidazole and for at least 24 hours after the completion of therapy.
   d. Treatment is recommended for symptomatic pregnant women: metronidazole 500 mg orally bid × 7 days.
   e. 90% of men with this infection are asymptomatic; therefore, sexual partners must be treated if reinfection is to be prevented.
5. Complications
   a. Can produce inflammatory changes on pap smear
   b. Has been associated with increased risk of premature rupture of membranes, preterm delivery, and postpartum endometritis

B. Vulvovaginal candidiasis

1. Pathophysiology
   a. Normal vaginal flora in up to 20% of healthy women
   b. Colonization or overgrowth of the organism (*Candida albicans*) can be caused by:
      (1) Diabetes mellitus
      (2) Pregnancy and postmenopause

(3) Drugs (oral contraceptives, antibiotics, steroids)
(4) Menstruation

2. Clinical presentation
   a. Extreme itching
   b. A thin watery to thick white discharge sometimes associated with dysuria or dyspareunia
   c. Physical examination: thick, white "cottage cheese" discharge, vulvovaginal erythema, and edema; may have satellite lesions on perineum
3. Diagnostic evaluation
   a. Pseudohyphae and spores may be seen on 10% potassium hydroxide preparation
   b. pH of vaginal secretions is normal (≤4.5).
   c. Latex agglutination testing is available for point-of-care testing.
4. Classification of vulvovaginal candidiasis
   a. Uncomplicated: 90% of cases
      (1) Sporadic/infrequent
      (2) Mild to moderate
      (3) Likely to be *C albicans*
      (4) Nonimmunocompromised patients
   b. Complicated
      (1) Recurrent or severe (≥4 episodes per year)
      (2) Candidiasis not caused by *C albicans*
      (3) Pregnancy
      (4) Debilitated, diabetic, or immunocompromised patients
5. Management
   a. Uncomplicated vulvovaginal candidiasis
      (1) Fluconazole 150 mg orally in one dose or
      (2) One of the following vaginal preparations every night × 3 days
         (a) Butoconazole 2% cream
         (b) Clotrimazole 2 (100 mg) vaginal tablets
         (c) Miconazole 200 mg vaginal suppository
         (d) Terconazole 80 mg suppository or 0.8% cream
   b. Complicated vulvovaginal candidiasis: use one of the following vaginal preparations every night × 7 days:
      (1) Butoconazole 2% cream
      (2) Clotrimazole 100 mg vaginal tablet
      (3) Miconazole 100 mg vaginal suppository
      (4) Terconazole 0.4% cream
      (5) Fluconazole 150 mg orally × 3 doses on days 1, 4, and 7

C. Bacterial vaginosis (most common cause of vulvovaginitis)

1. Pathophysiology
   a. Previously thought to be caused by a single organism, it is now evident that multiple anaerobes are implicated in the pathogenesis of this vaginal infection.

b. It is believed to result from changes in the vaginal microflora that foster the synergistic activity of the normal mixed flora and anaerobes, which ultimately results in the predominance of the anaerobic organisms.

2. Clinical presentation: patients present with complaints of a fishy smelling discharge and mild itching.
3. Diagnostic criteria: three of the following must be present:
   a. Homogeneous, grey-white, noninflammatory discharge that coats the vaginal wall rather than pooling on the vaginal floor
   b. Positive amine odor test (fishy odor when potassium hydroxide is added to a sample of the vaginal secretions)
   c. pH of vaginal secretions >4.5
   d. "Clue cells" (vaginal epithelial cells covered with bacteria) on wet mount
   e. DNA probe-based test available for point-of-care testing
4. Management
   a. Metronidazole 500 mg orally bid × 7 days
   b. Alternative regimens
      (1) Clindamycin cream 2% intravaginally 5 g at bedtime × 7 days or
      (2) Metronidazole gel 0.75% intravaginally 5 g/day × 5 days or
      (3) Clindamycin 300 mg orally bid × 7 days
      (4) Tinidazole 2 g/day orally × 2 days
      (5) Tinidazole 1 g/day orally for 5 days
   c. Treatment with oral metronidazole or clindamycin is recommended in symptomatic pregnant women.
5. Complications: in pregnant patients, bacterial vaginosis is associated with an increased incidence of premature rupture of membranes, preterm labor, preterm birth, and endometritis after cesarean section.

II. CERVICITIS

A. Etiology

1. *Chlamydia*
2. Gonorrhea
3. *Trichomonas*

B. Clinical presentation

1. Vaginal discharge and dysuria
2. May be asymptomatic
3. Physical examination

C. Diagnostic evaluation

1. Test for gonococcal cervicitis and *Chlamydia* with either nucleic acid testing or culture.
   a. Vulvovaginal area appears normal.
   b. Cervix is friable and inflamed with a purulent endocervical discharge.
2. On wet mount and Gram stain, look for WBCs, trichomonads, and gonococcal cervicitis; if only WBCs are seen, assume *Chlamydia*.

D. Management

1. Trichomonal cervicitis is treated with metronidazole 2 g orally as a single dose or 500 mg orally bid × 7 days.
2. Gonococcal cervicitis is treated with:
   a. First line: ceftriaxone 250 mg IM in a single dose
   b. Second line
      (1) Azithromycin 2 g orally or
      (2) Cefixime 400 mg orally in a single dose
   c. Because of increasing resistance, fluoroquinolones are no longer recommended for treatment of gonorrhea.
3. Because concurrent infection with *Chlamydia trachomatis* is common in these patients, a regimen effective against *Chlamydia* (described below) should also be prescribed.
4. Chlamydial cervicitis should be suspected if the Gram stain reveals PMNs but no organisms. It is treated with:
   a. Azithromycin 1 g orally in a single dose (the preferred treatment) or
   b. Doxycycline 100 mg orally bid × 7 days
   c. Alternative regimens
      (1) Ofloxacin 300 mg orally bid × 7 days or
      (2) Erythromycin base 500 mg orally qid × 7 days or amoxicillin 500 mg orally tid × 7 days (recommended for pregnant women) or
      (3) Erythromycin ethylsuccinate 800 mg orally qid × 7 days or
      (4) Levofloxacin 500 mg orally × 7 days
5. Recommended treatment for pregnant women
   a. Azithromycin l g orally in a single dose
   b. Amoxicillin 500 mg orally tid × 7 days; alternative is erythromycin base 500 mg orally qid × 7 days.
6. Contraindicated in pregnant or lactating women and in children/adolescents
   a. Ciprofloxacin and ofloxacin are contraindicated during pregnancy and in patients <17 years old.
   b. Doxycycline is contraindicated in pregnant/lactating women and in children <8 years old.
   c. Erythromycin estolate is contraindicated in pregnancy.

III. MISCELLANEOUS VULVOVAGINAL CONDITIONS

A. Contact vulvovaginitis

1. Clinical presentation
   a. Itching and irritation with erythema and edema
   b. No discharge
2. Evaluation for an infectious etiology is negative.
3. Management
   a. Determine the cause (cosmetics, toiletries, laundry detergent) and eliminate it.
   b. Cool sitz baths and wet compresses of Burow's solution will suffice in mild to moderate cases; topical steroids are indicated for severe cases.

B. Vaginal foreign body

1. Children may insert any object; adults may forget tampons, pessaries, diaphragms, or condoms.
2. Clinical presentation: a foul-smelling and sometimes bloody discharge
3. Management
   a. Remove the object.
   b. Treat for any vaginitis and arrange for follow-up.
4. Consider the possibility of sexual abuse when vaginal foreign bodies are found in prepubertal girls.

C. Bartholin abscess

1. Etiology: Bartholin gland or duct cyst can become infected with various organisms (*Escherichia coli, N gonorrhoeae, Chlamydia,* or mixed anaerobes)
2. Clinical presentation
   a. Painful unilateral swelling of the posterior-lateral aspect of the vaginal opening
   b. Pain is increased with sitting and walking; associated swelling and erythema of the labia majora is common.
3. Management
   a. Incision and drainage (on the mucosal aspect of the vestibule) followed by sitz baths
   b. Antibiotics if cellulitis is also present
   c. Word catheter placement for 6–8 weeks (to facilitate healing and reepithelialization)
   d. Elective marsupialization

IV. PELVIC INFLAMMATORY DISEASE

A. Etiology

1. **Usually a sexually transmitted infection**
2. **Most common cause is ascension from asymptomatic cervicitis**
3. **Risk factors**
   a. **Multiple sexual partners**
   b. **Previous pelvic inflammatory disease**
   c. **Adolescence**
   d. **Use of an intrauterine device use (especially during the first few months of use)**
   e. **Instrumentation of the cervix or uterine cavity**
   f. **Recent menses**
   g. **Douching**
   h. **Cigarette smoking**
4. **50% of patients have no identifiable risk factors.**
5. **Pathogenic organisms: *N gonorrhoeae* and *Chlamydia trachomatis* are the major pathogens; together they account for approximately 80% of cases.**
   a. ***Chlamydia* (25%–50% of cases)**
      (1) **Most common cause of pelvic inflammatory disease in college students**

(2) An obligate intracellular parasite that penetrates the submucosa and muscularis layers, producing a more indolent infection than gonorrhea

(3) These infections may occur at any time in the menstrual cycle and have the greatest propensity for causing permanent tubal obstruction and infertility.

b. *N gonorrhoeae* (25%–50% of cases)

(1) Most common cause of pelvic inflammatory disease in urban areas

(2) A gram-negative intracellular diplococcus

(3) Virulence is related to the ability to attach to tubal epithelial cells; infection is usually limited to the mucosal surface.

(4) These infections generally develop shortly after menses.

c. Other pathogens

(1) Anaerobic bacteria

(2) Aerobic bacteria

(3) Genital tract mycoplasmas

B. Pathophysiology

1. Normal host barriers (uterotubal cervical mucus, lysozymes, and local immunoglobulin IgA as well as the cervix itself) usually prevent infection.
2. Menstruation is the most common cause of breakdown of these barriers.
3. During pregnancy, fusion of the chorion and decidua forms another natural barrier; it obliterates the cavity between the cervix and the fallopian tubes, making pelvic inflammatory disease during pregnancy a less common event.

C. Diagnostic evaluation

1. Laboratory studies
   a. Routine laboratory studies (CBC, sedimentation rate) may be supportive but are nonspecific.
      (1) Leukocytosis (WBC count >10,500/mm^3) supports the diagnosis but may not be present.
      (2) The sedimentation rate is increased (>15 mm/hr) in most cases.
   b. Gram stain of endocervical specimen (98% specific but only 67% sensitive for gram-negative intracellular diplococci)
   c. Gonorrhea nucleic acid testing or culture
   d. Chlamydial nucleic acid testing or culture
   e. Measurement of beta-human chorionic gonadotropin (ß-HCG) is useful in excluding an ectopic pregnancy.
2. Laparoscopy is the most accurate way to diagnose pelvic inflammatory disease. Although neither practical nor necessary for most cases, it should be considered for equivocal or refractory cases. It allows:
   a. Direct visualization and culture of the involved organs
   b. Exclusion of other diagnoses (appendicitis, diverticular disease)
3. Ultrasonography may diagnose tubo-ovarian abscess or pyosalpinx.

D. Criteria for diagnosing acute pelvic inflammatory disease

1. CDC recommends empirical treatment for pelvic inflammatory disease in sexually active women with pelvic pain plus one of the following:

a. Uterine tenderness
b. Adnexal tenderness (usually bilateral)
c. Cervical motion tenderness: any pain more than patient's expectation

2. Additional criteria helpful in increasing the specificity but not required for the diagnosis
   a. Routine criteria
      (1) Oral temperature >101°F (38.3°C)
      (2) Abnormal vaginal or cervical discharge
      (3) Increased erythrocyte sedimentation rate or C-reactive protein
      (4) Laboratory evidence of cervical infection with *N gonorrhoeae* or *Chlamydia*
      (5) WBC count >10,500/mm^3
   b. Definitive criteria
      (1) Confirmation of fluid-filled tubes (with or without free pelvic fluid) or a tubo-ovarian abscess on ultrasonography
      (2) Histopathologic confirmation of endometritis on endometrial biopsy
      (3) Findings consistent with pelvic inflammatory disease on laparoscopic examination

E. Management

1. All regimens should cover *N gonorrhoeae*, *Chlamydia*, anaerobes, gramnegative rods, and streptococci.
2. Outpatient: ceftriaxone 250 mg IM or another parenteral third-generation cephalosporin, or cefoxitin 2 g IM with probenecid 1g orally plus doxycycline 100 mg orally bid × 14 days is the traditional therapy, ± metronidazole 500 mg orally bid × 14 days.
3. Inpatient
   a. Indications for admission
      (1) Toxic patient
      (2) Nausea and vomiting, unable to take antibiotics
      (3) No response to outpatient therapy within 72 hours
      (4) Suspicion of a tubo-ovarian or pelvic abscess (which may require surgery)
      (5) Concurrent pregnancy
      (6) Concurrent HIV infection
      (7) Uncertain diagnosis (another surgical emergency cannot be excluded)
      (8) Young age (potential for future fertility issues)
      (9) Timely clinical follow-up (within 72 hours of starting antibiotics) cannot be arranged
   b. Drug therapy
      (1) Cefoxitin 2 g IV every 6 hours or cefotetan 2 g IV every 12 hours plus doxycycline 100 mg IV or orally bid. Continue this regimen for at least 24 hours after clinical improvement; after discharge, patients are continued on doxycycline 100 mg orally bid for a total of 14 days. When tuboovarian abscess is present, metronidazole or clindamycin is added to doxycycline for oral therapy.
      (2) An alternative regimen is clindamycin 900 mg IV every 8 hours plus gentamicin: loading dosage 2 mg/kg IV or IM followed by a maintenance dosage of 1.5 mg/kg every 8 hours. This regimen is continued for at least 24 hours after signs of improvement. Single daily dosing (3–5 mg/kg) can be substituted. After discharge, continue doxycycline 100 mg orally every 12 hours or clindamycin 450 mg orally

every 6 hours for a total course of 14 days. Clindamycin is preferred when tubo-ovarian abscess is present.

(3) Ampicillin/sulbactam 3 g IV every 6 hours plus doxycycline 100 mg orally or IV every 12 hours

F. Complications

1. Pelvic inflammatory disease is a significant cause of ectopic pregnancy, infertility, and chronic pelvic pain.
2. Formation of tubo-ovarian abscess
3. Fitz-Hugh-Curtis syndrome (perihepatitis)
4. Obstetrical complications
   a. Intrauterine growth retardation
   b. Septic abortion
   c. Premature rupture of membranes/preterm delivery
5. Sexual partners should be evaluated and treated empirically to prevent reinfection (if they had sexual contact within 60 days of symptom onset).

V. OVARIAN DISORDERS

A. Adnexal torsion

1. Pathophysiology
   a. An abnormal ovary or fallopian tube twists around its vascular pedicle → blood supply is compromised → painful anoxic degeneration of the ovary or fallopian tube with eventual gangrenous necrosis
   b. Ovarian torsion usually occurs in association with an ovary that is enlarged because of a cyst or tumor or that is overstimulated (ovarian hyperstimulation syndrome) by use of fertility drugs; fallopian tube torsion usually occurs in association with hydrosalpinx, neoplasm, adhesions, trauma, or previous ligation.
   c. May occur at any age (although most common in the mid-twenties) and at any time during the menstrual cycle
2. Clinical presentation
   a. Vital signs: absence of fever, or tachycardia out of proportion to the fever (different from pelvic inflammatory disease, tubo-ovarian abscess)
   b. History
      (1) Onset of pain is usually sudden (frequently during or immediately after sexual intercourse); the patient may even remember the exact moment. Pelvic infections have a more gradual onset.
      (2) The pain is usually unilateral (more commonly on the right), becomes increasingly severe but may subside and, if so, the patient may not appear to be acutely ill on presentation (not so with pelvic infections).
      (3) Often there is a history of similar episodes in the past that resolved spontaneously.
   c. Pelvic examination
      (1) While cervical motion tenderness may be present, unilateral adnexal tenderness is the rule (although bilateral adnexal tenderness is found in some patients).

(2) A significant finding is a small, discrete, tender adnexal mass. Inflammatory masses tend to be larger and less well defined.

3. Diagnostic evaluation
   a. Routine laboratory studies are usually normal. Pregnancy testing should be done to exclude ectopic or concomitant intrauterine pregnancy.
   b. Ultrasound may reveal the adnexal mass when the physical examination does not. Color Doppler flow aids diagnosis. Coiled vessels or "whirlpool sign" is 88% accurate in diagnosing torsion.
   c. CT/MRI: thickened fallopian tube, enlarged ovary, ovarian mass, free pelvic fluid, edematous ovary, deviation of the uterus to the affected side, associated hemorrhage
   d. Laparoscopy: the definitive diagnostic procedure
   e. Negative imaging findings should be taken with caution when clinical suspicion of torsion is high.
4. Management
   a. Surgical correction or resection is the definitive therapy of choice; however, laparoscopy alone is being used with increasing frequency, because "untwisting" may be possible if torsion is diagnosed early or is caused by the ovarian hyperstimulation syndrome.
   b. While return of ovarian function is possible even with late presentation, pediatric patients taken to surgery within 8 hours had the best chance of recovery.
5. Complications
   a. Scarred tube → ↓ fertility and ↑ risk of tubal pregnancy
   b. Ovarian necrosis → peritonitis and shock

B. Hemorrhagic corpus luteum cyst

1. Etiology
   a. Corpus luteum of pregnancy persists up to 8 weeks into the pregnancy.
   b. Persistent corpus luteum in a nonconception cycle may occur and cause amenorrhea until it degenerates; this is followed by heavy menstrual bleeding.
2. Classic clinical scenario
   a. May be indistinguishable from a ruptured ectopic pregnancy
   b. Sudden onset of lower abdominal pain in an amenorrheic patient or one in early pregnancy may be the only clue. The diagnosis is easier to make in pregnant patients, because the serum pregnancy test will be positive and ultrasound will show intrauterine pregnancy. (The likelihood of a coexisting normal and ectopic pregnancy is exceedingly rare, unless the gestation was achieved through assisted reproductive technology.) In the nonpregnant patient, a negative ß-HCG and an adnexal mass coupled with a missed period or two can be easily interpreted as an ectopic pregnancy; ultrasound will help sort this out.
   c. Laparoscopy is necessary for definitive diagnosis as well as to establish if cystic bleeding has stopped.
3. Management
   a. Surgical (unless cystic bleeding has stopped and the patient is stable)
   b. Usually requires ovarian cystectomy

C. Mittelschmerz

1. Etiology
   a. Pain associated with rupture of a follicular cyst and extrusion of an ovum from the ovary
   b. The pain is believed to be due to follicular fluid irritation of the parovarian visceral peritoneum.
2. Clinical presentation
   a. Occurs at midcycle (days 12–16 in women with 28- to 30-day cycles) after a normal menses
   b. The pain is unilateral, varies in intensity, lasts from a few hours to a day, and may be associated with slight vaginal spotting.
3. Management: supportive with analgesics

VI. UTERINE DISORDERS

A. Abnormal vaginal bleeding (nonpregnancy-related)

1. Etiology
   a. Alterations in the endocrine system secondary to:
      (1) Ovarian tumors
      (2) Corpus luteum cyst
      (3) Menarche or menopause
      (4) Excessive exercise
      (5) Poor diet
   b. Drugs
      (1) The most common drugs implicated in "breakthrough" bleeding (ie, bleeding that occurs between periods)
         (a) Anticonvulsants (eg, phenytoin, phenobarbital, carbamazepine)
         (b) Some antibiotics (penicillins, tetracyclines, and trimethoprim-sulfamethoxazole)
      (2) Other pharmaceutical causes of abnormal bleeding
         (a) Anticoagulants
         (b) Exogenous estrogen or progesterone
         (c) Oral contraceptives (→ breakthrough and withdrawal bleeding)
   c. Pelvic infections
      (1) Vulvovaginitis
      (2) Cervicitis
      (3) Endometritis
      (4) Pelvic inflammatory disease
   d. Neoplasms (of both external and internal genitalia)
      (1) Cervical polyps or carcinoma (bleeding often provoked by coitus)
      (2) Leiomyomas (fibroids)
      (3) Endometrial polyps or carcinoma
      (4) Ovarian tumors

e. Postoperative (generally occurs when absorbable sutures dissolve)
f. Trauma
   (1) Foreign bodies
   (2) "Straddle" injury
g. Intrauterine devices
h. Medical problems
   (1) Coagulopathies (especially von Willebrand disease, the most common hereditary disorder associated with menorrhagia)
   (2) Thrombocytopenia
   (3) Hypothyroidism
   (4) Cirrhosis
i. Dysfunctional uterine bleeding

2. Management
   a. Exclude life-threatening hemorrhage by evaluating hemodynamic stability (orthostatic vital signs, hemoglobin/hematocrit).
   b. Stabilize patients with life-threatening bleeding (the minority) with IV fluids and blood products as needed. Consider IV estrogens.
   c. Identify treatable causes through a careful history and physical; important points include:
      (1) Assume that any woman of childbearing age with abnormal vaginal bleeding is pregnant (intrauterine or ectopic) until proven otherwise; check a pregnancy test.
      (2) Estimate the blood loss by determining the duration and amount of bleeding as well as the presence of clots (a history of heavy bleeding associated with clotting >7 days indicates substantial blood loss).
      (3) In addition to performing a pelvic examination, check other potential sites of bleeding that might be confused with vaginal bleeding (perineum and rectum); also, check for bladder tenderness and order a urinalysis if there is any question as to the source of the bleeding.
   d. Order other appropriate laboratory studies as indicated.
   e. Iron-deficiency anemia suggests prior excessive bleeding in the absence of severe bleeding on physical examination; assessment of the hemoglobin level (plus microscopic examination of a peripheral smear) or a serum iron (ferritin) concentration is a clinically useful means of finding iron-deficiency anemia.
   f. Cervical bleeding
      (1) Mild to moderate → Monsel solution or silver nitrate
      (2) Severe (postoperative or trauma-induced) → appropriate suturing
   g. Refer to a gynecologist for a complete investigation (including an endometrial biopsy if the patient is >35 years old).

B. Gestational trophoblastic disease

1. Group of pregnancy-related tumors
2. Overgrowth of molar pregnancies and or abnormal placental cells; 2%–3% develop into choriocarcinoma and may be malignant.
3. Typical presentation of pregnant woman with hyperemesis, high ß-HCG early in pregnancy and passage of hydropic vesicles ("bunch of grapes") or single grapelike tissue from the vagina

4. Ultrasound shows a snowstorm pattern.
5. Treatment requires uterine evacuation and chemotherapy.

C. Leiomyoma

1. Fibroid uterus is a common benign muscular tumor of the uterus.
2. Incidence increases with age; affects >40% of women by age 35.
3. Black American women are more likely to have fibroids.
4. May cause anemia secondary to significant bleeding or become painful when large enough to become necrotic
5. Diagnostic evaluation: ultrasound examination; however, the enlarged uterus may be palpable on bimanual examination.
6. Management
   a. NSAIDs to control pain
   b. Iron supplementation and potentially blood transfusion for anemia
   c. Hysterectomy, myomectomy, or embolization if indicated

VII. SEXUAL ASSAULT

A. General approach

1. **Purpose of evaluation is to diagnose and treat the victim's physical and emotional injuries, and to collect legal evidence.**
2. **Because the patient was a victim of a violent crime, she may feel fearful, helpless, and guilty. For this reason, the interview and examination should be performed patiently in a quiet, private area and by the same doctor-nurse-counselor team from start to finish (multidisciplinary teams recommended). Offer to call the crisis counselor immediately to be with the patient during the interview and examination, as well as arrange for follow-up care at a rape crisis center. Follow state and local protocols. Specialized, trained nurses are commonly used for this examination.**
3. **Explain all procedures and obtain consent for the physical examination, evidence collection, photographs, and treatment.**
4. **Divide the history taking into two segments: a standard medical history (to determine the need for medical evaluation and treatment) and a history of the assault (to correlate with the physical findings). Group history taking of the assault victim saves time and avoids inconsistencies or contradictions.**

B. History of the event

1. **Where and when did the assault occur?**
2. **What happened during the assault? Use the patient's own words with quotations and ask about the following:**
   a. **Number of assailants**
   b. **Use of force, weapons, objects, or restraints**
   c. **Orifices penetrated and an impression of whether or not ejaculation occurred**
   d. **Use of alcohol or drugs**
3. **What happened after the assault?**
   a. **Did the patient bathe, void, defecate, brush teeth, or change clothes?**

b. Does she complain of pain or have any other symptoms?

4. Has the patient had sexual intercourse in the last 72 hours? (If so, it may confuse the laboratory analysis of sperm and acid phosphatase.)
5. Determine pregnancy risk.
   a. When was the last menstrual period (midcycle is high risk)?
   b. Is the patient using any birth control method?

C. Physical examination

1. Note the general appearance of the patient as well as her clothing.
2. Perform a general physical examination, noting especially any signs of trauma.
3. Perform a pelvic examination, looking for evidence of trauma or infection. In many cases, there is no visible trauma despite penetration.

D. Laboratory and evidence collection

1. The following tests are medically indicated:
   a. Standard laboratory studies and radiographs when indicated for trauma
   b. ß-HCG (to exclude preexisting pregnancy)
2. Evidence
   a. Know what the police want before you examine the patient, or use a standard kit that contains supplies for the collection of any or all of the following:
      (1) Debris from skin
      (2) Dried secretions on skin
      (3) Combed and plucked head and pubic hairs
      (4) Fingernail scrapings and clipped fingernails
      (5) Saliva sample
      (6) Oral, anal, and vaginal smears (for sperm and acid phosphatase)
      (7) Blood sample
      (8) Nasal mucus sample
   b. Photographs are becoming increasingly important. Develop protocol for forensic admissibility and protect the victim's privacy.
   c. The emergency physician should personally collect and examine a wet mount for motile sperm. Sperm motility is a good sign of recent intercourse. If you wait for the pathologist to examine the specimen, sperm motility may be lost. Remember, however, that penetration (not ejaculation) is the hallmark of rape.
   d. Maintain an unbroken chain of evidence.
      (1) Specimens must always be in view or under lock and key.
      (2) Sign the outside of each sealed container when transferring custody.

E. Management

1. Standard care for traumatic injuries
2. Prevent infection
   a. Offer prophylactic antimicrobial therapy for syphilis, gonorrhea, *Chlamydia*, *Trichomonas*, and bacterial vaginosis.
   b. Offer prophylaxis for hepatitis B with vaccination.

c. Although HIV seroconversion is in general a low risk with sexual assault, any high-risk circumstances such as anal penetration, obvious trauma, or known HIV infection in the assailant should be treated prophylactically. Some authors advocate offering prophylaxis to all sexual assault victims within 72 hours. Current treatment recommendations: combination therapy with tenofovir plus emtricitabine (1 tab orally once a day for 28 days) or lamivudine plus zidovudine (1 tab orally twice a day for 28 days); if higher risk, consider adding lopinavir plus ritonavir (400 mg of lopinavir and 100 mg of ritonavir [two tabs] orally twice a day for 28 days).

d. Schedule follow-up visits for repeat serology and cultures.

3. Prevent pregnancy: offer postcoital contraception (levonorgestrel 1.5 mg once); refer for repeat pregnancy test as needed.
4. Counseling: The initial contact should be made while the patient is in the emergency department by an individual experienced in rape counseling; if this is not possible, early referral is essential. The postexposure prophylaxis hotline is 888-448-4911.

OBSTETRIC DISORDERS

I. DIAGNOSIS OF PREGNANCY

ANY WOMAN OF CHILDBEARING AGE WHO PRESENTS TO THE EMERGENCY DEPARTMENT IS PREGNANT UNTIL PROVEN OTHERWISE.

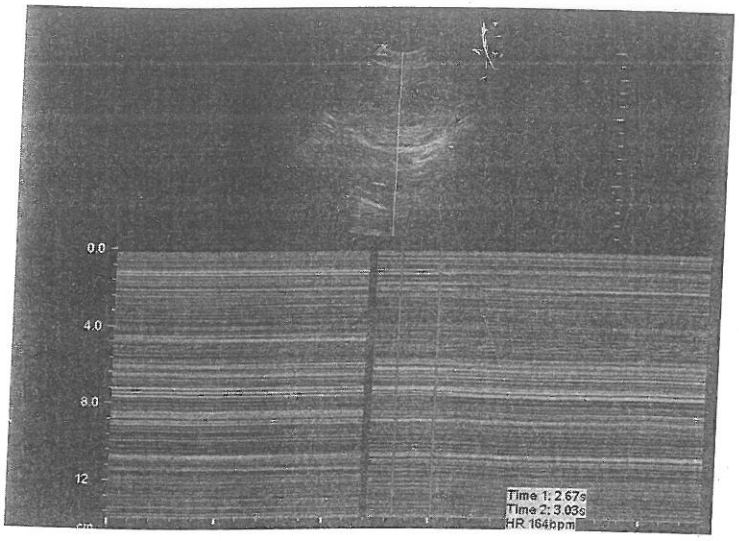
A. Clinical presentation

1. Menstrual period: missed, light, or late
2. Breast swelling and tenderness at 4 weeks
3. Fatigue, nausea, and urinary frequency at 5–6 weeks
4. Cervical softening (tip at 4 weeks, isthmus at 6–8 weeks)
5. Chadwick sign (bluish discoloration of cervix at 6–8 weeks)
6. Uterus enlarged and soft at 6–8 weeks

B. Pregnancy tests

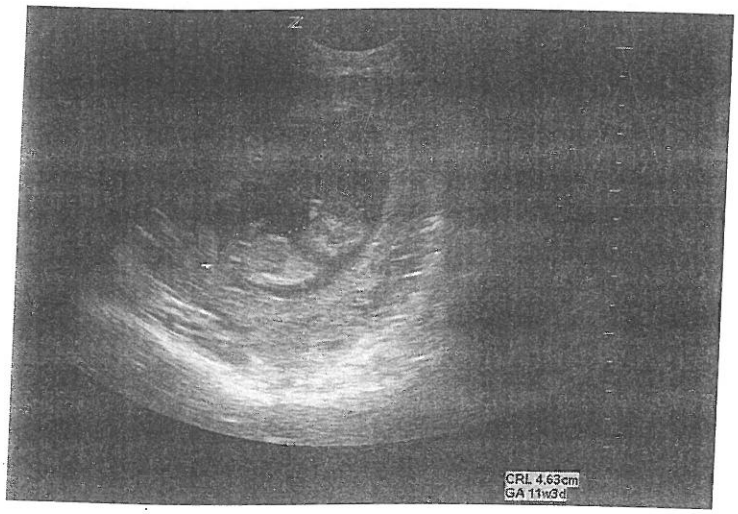
1. All available tests detect ß-HCG with varying sensitivities and specificities. Production of ß-HCG begins at the time of implantation (8–9 days after conception). Levels of ß-HCG double every 2 days during the initial weeks of a normal pregnancy, peak at 8 weeks, and remain detectable up to 60 days after birth or abortion. With the newer assays, pregnancy can be detected as early as 23–25 days after the last menstrual period. Pregnancy tests that measure the ß-HCG subunit have the greatest specificity. ELISA is the standard assay. Urine ELISA has a sensitivity for levels of ß-HCG between 25 and 50 mLv/mL. Qualitative serum ELISA has a sensitivity of 10 mLv/mL. Quantitative serum ELISA has a sensitivity of 5 mLv/mL. You need to be aware of the sensitivity and specificity of the pregnancy assay being used by the laboratory and the reference standard being used (first, second, or third international reference preparation) to be able to correctly interpret the test result.
2. Causes of false negatives
   a. Too early in pregnancy
   b. Dilute or old urine: if the specific gravity is below the recommended level set by your laboratory, get a serum qualitative.
   c. Ectopic pregnancy: although rare, ruptured ectopic pregnancies have been reported with very low or absent ß-HCG.
   d. Incomplete abortion
3. Causes of false positives (rare)
   a. Interference from non–ß-HCG substances
      (1) Human luteinizing hormone
      (2) Rheumatoid factor
      (3) Heterophile Ab
      (4) Anti-animal Ab
      (5) Pituitary HCG
   b. Exogenous HCG
   c. Trophoblastic neoplasm
4. Urine ELISA testing (rather than serum testing) reduces the incidence of false-positive and false-negative results, because this assay is not affected by drugs or physiologic abnormalities.

5. Ultrasound is frequently used in the emergency department to confirm that the pregnancy is intrauterine and to verify fetal heart rate.
   a. Fetal heart can be determined by using M-mode on ultrasound.
   b. Fetal age can be determined by measuring the gestational sac, the crown–rump length, the head circumference, or the femur length. The best measurement depends on the age and position of the fetus.
   c. Ultrasound should be performed only by trained personnel.



Courtesy of Sarah Sommerkamp, MD

M-mode ultrasound showing measurement of fetal heart rate



Courtesy of Sarah Sommerkamp, MD

Ultrasound showing crown–rump length measurement for gestational age

C. Physiologic changes of pregnancy

1. Cardiovascular changes
   a. Heart rate increases by 15–20 beats per minute above baseline (to an average of 80–90 beats per minute) by the third trimester.
   b. Blood pressure

(1) Systolic and diastolic blood pressure decrease by 10–15 mmHg in the second trimester (to an average of 102/55 mmHg) and then gradually return to prepregnant levels in the third trimester.

(2) After the 20th week of gestation, uterine compression of the inferior vena cava in the supine position can decrease venous return by as much as 30% and produce hypotension. This can be relieved by placing the patient in the left lateral decubitus position, inserting a wedge under the right hip, or manually displacing the uterus.

c. Cardiac output increases by ~30% during the first trimester, increases further (~50%) in the second trimester and is maintained at this level for the remainder of the pregnancy.

2. Respiratory changes
   a. Respiratory rate increases slightly (or not at all).
   b. Tidal volume increases by 40%.
   c. Functional residual volume decreases by 25% (due to elevation of the diaphragm).
   d. pCO_2 decreases to 30–33 mmHg.
3. Hematologic changes
   a. Blood volume increases up to 50% at term
   b. Hematocrit decreases to the low 30% range by the 30th week.
   c. WBC count increases (due to an increase in PMNs); counts up to 18,000/mm^3 are normal in the second and third trimesters, while counts up to 25,000/mm^3 may be seen during labor.
   d. Erythrocyte sedimentation rate is increased to 70–80 mm/hr.
   e. Fibrinogen level is increased (average level is 400–450 mg/dL).
   f. Prothrombin time (INR)/partial thromboplastin time are normal despite increases in factors VII, VIII, IX, X, and XII.

II. DRUGS AND RADIATION EXPOSURE IN PREGNANCY

A. Drugs

1. In the eyes of an attorney, all drugs are harmful and, because adverse maternal/fetal effects are unknown for many of them, they should be used only when absolutely indicated for maternal or fetal health.
2. The teratogenic risk associated with drugs is greatest during embryogenesis (the first trimester) and is usually dose-related.
3. The FDA classifies drugs used in pregnancy drugs into five categories (A, B, C, D, X) according to their safety:
   a. Drugs in category A have been studied in people and are considered safe for use in pregnancy.
   b. Drugs in category B have been studied in animals and are generally considered safe.
   c. Drugs in category C should be used with caution.
   d. Drugs in categories D and X should be avoided (if possible); studies have demonstrated use in pregnancy associated with risk to the fetus.
4. Drugs that are generally recognized as safe
   a. Penicillins
   b. Cephalosporins

c. Erythromycin (except the estolate)
d. Azithromycin
e. Aztreonam
f. Spectinomycin
g. Acetaminophen
h. Narcotics (oxycodone, hydromorphine)
i. Heparin
j. Asthma drugs
   (1) Inhaled ß-adrenergic agents (especially terbutaline)
   (2) Steroids
   (3) Theophylline (a category C drug)
   (4) Inhaled anticholinergic agents
   (5) Cromolyn
k. Antiemetics if dehydration or poor maternal nutrition is a threat; ondansetron and metoclopramide are the drugs of choice (category B), followed by trimethobenzamide, promethazine, and prochlorperazine (category C).

5. Immunizations
   a. Those derived from killed viruses (tetanus, diphtheria, hepatitis B, rabies) can be safely administered in pregnancy.
   b. Those derived from live attenuated viruses (measles, mumps, rubella, smallpox) are contraindicated in pregnancy.

B. Radiation exposure

1. The risk of birth defects from a 1-rad exposure (1,000 mrads) is thousands of times smaller than the spontaneous risks of congenital malformations or genetic disease. If exposure to the fetus is <5–10 rads, there is no significant increase in birth defects; also, coned radiographic beams aimed >10 cm away from the fetus are not harmful.
   a. The initial trauma series (plain radiographs of cervical spine, chest, and pelvis) all deliver <1 rad.
   b. CT scanning exposes the fetus to a higher level of radiation, which can be reduced by shielding the uterus for part of the study. With shielding, fetal exposure from:
      (1) Head and chest scans is <1 rad
      (2) Abdominal scan (above the uterus) is <3 rad
      (3) Pelvic scans (centered over the fetus) is <3 rads
      (4) Spiral scanning can reduce the dosage even further (up to 30%).
   c. MRI scanners do not use radiation and produce no birth defects but often are not available on a 24-hour basis and may be inappropriate for unstable patients.
2. Clinical guidelines
   a. Do not withhold necessary radiographic studies; shield the uterus, and limit the scope of the examination when possible.
   b. If the patient is not sterilized (or is not using another reliable contraceptive method), postpone elective radiographs until a negative urine pregnancy test is obtained or until after the first 10 days after a normal period.

III. MEDICAL COMPLICATIONS OF PREGNANCY

A. Nausea and vomiting

1. 50%–70% of women are affected, usually in the first trimester (probably due to increased ß-HCG levels). Some studies suggest a vitamin B_6 deficiency as the cause and recommend supplemental vitamin B_6.) Persistence of symptoms past 14 weeks or presence of abdominal pain suggests the possibility of another cause.
2. Management
   a. Frequent small meals (dry crackers before rising, toast and cereal when symptomatic) and supplemental vitamin B_6 may be helpful.
   b. IV hydration (D5 lactated Ringer's or D5 normal saline) and antiemetics are indicated if ketosis or hyperemesis gravidarum develops (starvation, dehydration, acidosis).
   c. Hospitalization is indicated if emesis cannot be controlled with the above measures.

B. Urinary tract infection and pyelonephritis

1. Pregnant women are more prone to these infections (and their associated complications) than nonpregnant women. The increased susceptibility is related to the following physiologic changes, which are induced by the gravid state:
   a. Increased mechanical pressure on the ureters and bladder by the uterus
   b. Poor emptying of the bladder with voiding
   c. Inhibition of ureteral peristalsis and dilatation by progesteroneinduced smooth muscle relaxation
2. Management
   a. Both symptomatic and asymptomatic bacteriuria should be treated; untreated bacteriuria in pregnancy is associated with a 20%–40% risk of progression to pyelonephritis.
   b. Send urine for culture and place patients on a 7-day course of an antibiotic active against *Escherichia coli*, *Proteus*, and *Klebsiella*. (All urinary tract infections in pregnant women are considered "complicated" and should not be treated as simple cystitis with 3 days of antibiotics.) Resistance rates have increased. The following commonly used antibiotics eradicate bacteriuria in 70%–80% of patients:
      (1) Cephalosporins
      (2) Nitrofurantoin (not effective against *Proteus*)
      (3) Amoxicillin (drug of choice in third trimester) or amoxicillin-clavulanate
      (4) Sulfas (use should be restricted to the first and second trimesters; sulfas given in the last few weeks of pregnancy may contribute to hyperbilirubinemia and kernicterus in the newborn)
   c. Consider chlamydial vaginitis as the cause of the urinary tract symptoms if the bacteriuria proves to be culture negative; a single 1-gram dose of azithromycin is safe and effective.
   d. Admit pregnant patients with pyelonephritis to the hospital for IV antibiotics (a cephalosporin with or without an aminoglycoside) and hydration. These patients are at increased risk of developing preterm labor, bacteremia, and septic shock. Urine cultures should be sent and an obstetric consult obtained.

C. Thromboembolism

1. Pregnancy is a hypercoagulable state.
   a. The synthesis of coagulation factors VII, VIII, IX, X, and XII is increased.
   b. Venous distensibility is increased.
   c. Compression of the inferior vena cava by the enlarging uterus promotes venous stasis.
2. The risk of thromboembolism is 5–6 times greater in pregnant women than in nonpregnant women and is highest in the immediate postpartum period as well as those delivered by C-section.
3. Initial diagnostic studies of choice
   a. Doppler ultrasonography or impedance plethysmography to exclude deep-vein thrombosis (abnormal results should be confirmed with the patient on her left side)
   b. Chest radiograph (with shielding) to exclude other pulmonary diseases when considering the diagnosis of pulmonary embolism
   c. Arterial blood gases: an alveolar-arterial (A-a) oxygen gradient >20–25 mmHg is abnormal and suggestive of pulmonary embolism. When interpreting these studies, recall that the diaphragms are symmetrically elevated in late pregnancy, and that respiratory alkalosis with a slight increase in the A-a gradient is normal in pregnancy and due to progesterone-induced stimulation of the minute ventilation.
   d. Ventilation/perfusion scan or multidetector CT angiography (debatable which study is preferable): literature prefers CT angiography to exclude pulmonary embolism.
4. Treatment is with heparin (or low-molecular-weight heparin), which does not cross the placenta.
5. Warfarin is contraindicated in pregnancy.

D. TORCH infections: toxoplasmosis, other (syphilis), rubella syndrome, cytomegalovirus, herpes simplex infection

1. Toxoplasmosis (eye, ear, neurologic dysfunction, rash, prematurity)
   a. Parasitic disease caused by *Toxoplasma gondii*
   b. Linked with exposure to cats
   c. Treatment: spiramycin in pregnancy
2. Syphilis (fever, failure to thrive, saddle nose, rash)
   a. For details, see pages 565–567.
   b. Treatment: penicillin
   c. If treated inappropriately, ~50% fatal.
3. Rubella syndrome (cataracts, deafness, patent ductus arteriosus)
   a. Viral illness
   b. Order a pregnancy test in all women of childbearing age with rubella.
   c. If the pregnancy test is positive, the patient should be referred for genetic counseling.

Time of Maternal Infection	Fetal Infection
First month	50%
Second month	25%
Third month	10%

4. Cytomegalovirus (neurologic dysfunction, chorioretinitis, hepatosplenomegaly, rash, microcephaly)
   a. Viral illness; minimal maternal symptoms
   b. No specific treatment; prevention
5. Herpes simplex infection (eye, brain, and skin disease)
   a. For details, see pages 569–571.
   b. Women with primary mucous membrane infections in the third trimester have an increased risk of dissemination, which may be life-threatening.
   c. Most (if not all) of the complications associated with lesions during pregnancy can be avoided by taking antiviral medications in the third trimester. Famciclovir and valacyclovir are pregnancy category B.
   d. Neonatal infection can be contracted via passage through an infected birth canal and is associated with significant mortality (50%) and morbidity (particularly neurologic sequelae).
   e. Active infection at the time of delivery carries a 30%–40% risk of infecting the infant.
   f. The presence of active lesions in a patient who is in labor is an indication for cesarean section.
   g. Treatment with IV acyclovir for serious infections.

E. Intrahepatic cholestasis of pregnancy (idiopathic jaundice of pregnancy, pruritus gravidarum)

1. Presents as mild jaundice (bilirubin <5 mg/dL) and pruritus in the third trimester
2. Alkaline phosphatase is increased 7–10 times normal, and serum transaminase levels are normal or slightly increased.
3. Associated with increased risk of premature delivery and fetal compromise
4. Management
   a. On an outpatient basis if the diagnosis is clear
   b. Pruritus is treated with antihistamines, ursodeoxycholic acid, bile salts, and benzodiazepenes, with variable success.
   c. Resolves spontaneously with delivery
5. Recurrence with subsequent pregnancies is not uncommon.

F. Chronic and gestational hypertension

1. Chronic high blood pressure is ≥140/90 mmHg: can be exacerbated by pregnancy; identified before conception or before the 20th week of gestation and persists for >6 weeks postpartum
2. Gestational high blood pressure is ≥140/90 mmHg on two occasions in a patient who was normotensive before 20 weeks of gestation.
3. Management depends on severity of disease.
   a. Mild hypertension: monitoring below ~150/100 mmHg
   b. Moderate hypertension: above threshold or end-organ damage
   c. Start hypertensive treatments: antihypertensive medications are all safe in pregnancy except ACE inhibitors and angiotensin-receptor blockers. Diuretics are second-line therapy because of volume constriction.
4. Complications: Hypertension in pregnancy is associated with increased risk of superimposed preeclampsia, abruptio placentae, prematurity, intrauterine growth retardation, and stillbirth.

G. Preeclampsia

1. Definition: an increase in systolic or diastolic blood pressure that develops after the 20th to 24th week of pregnancy in a previously normotensive or hypertensive woman associated with proteinuria; patients with a history of chronic hypertension are more prone to develop preeclampsia and eclampsia.
2. Diagnosis

Table 23: Severity of Preeclampsia

Diagnosis	Mild	Severe
Increased blood pressure	Systolic ≥140 or ≥20 mmHg above baseline Diastolic ≥90 or ≥10 mmHg above baseline	≥160/110 mmHg
Proteinuria	0.3 g/24 hours	>5 g/24 hours
Edema (generalized)	Often but not always present	Present
Oliguria	Absent	Present
Associated symptoms		
Headache	Absent	Present
Visual disturbances	Absent	Present
Abdominal pain	Absent	Present
Pulmonary edema or cyanosis	Absent	Present
Associated laboratory findings		
Thrombocytopenia	Absent	Present
↑ Liver enzymes	Absent	Present
↑ Bilirubinemia	Absent	Present
↑ Serum creatinine	Absent	Present
↑ Uric acid	Usually present	Present
Intrauterine growth retardation	Absent	Present

3. Risk factors
   a. Primiparity: >60% of cases occur during the first pregnancy.
   b. Age: primigravidas at the extremes of age (<17 or >35 years old) are at increased risk.
   c. Pregnancies associated with a large placenta (multiple pregnancies, hydatidiform mole, diabetes mellitus)
   d. History of chronic hypertension or renal disease
   e. Family history of preeclampsia (mother, sister) or pregnancyinduced hypertension
4. Management
   a. If hypertension is the only finding:
      (1) Order a 24-hour urine for protein.
      (2) Encourage patient to decrease activity level, and recommend home blood pressure monitoring.
      (3) Refer patient to an obstetrician for close follow-up in 2–3 days.

b. If other findings are also present (especially proteinuria), hospitalization is indicated.
   (1) Order absolute bed rest with the patient lying on her left side.
   (2) Obtain a quantitative 24-hour urine for protein as well as renal function studies.
   (3) Observe for progression.

c. Antihypertensive therapy is not required unless the systolic blood pressure is >170 mmHg or the diastolic blood pressure is >105 mmHg; target pressure is 130–150 mmHg systolic and 90–100 mmHg diastolic.
   (1) Hydralazine is used most commonly, but other drugs work well, too (eg, diazoxide, labetalol).
   (2) ACE inhibitors (all the "-prils," eg, captopril, enalapril) are contraindicated because of the possibility of fetal renal damage.
   (3) Prophylactic treatment with magnesium is also recommended to prevent seizures in the preeclamptic patient (loading dose of 4–6 g over 30 minutes followed by a 2 g/hr drip to maintain a serum level of 4–7 mEq/L).

H. Eclampsia (preeclampsia and grand mal seizures or coma)

1. **Important facts**
   a. **Eclampsia may occur without prior proteinuria.**
   b. **Eclampsia can occur up to 28 days postpartum. One-third of eclamptic seizures occur after birth.**
   c. **Intracranial hemorrhage is the major cause of maternal death.**
2. **Warning signs of impending seizure**
   a. **Headache**
   b. **Visual disturbances**
   c. **Hyperreflexia**
   d. **Abdominal pain (epigastric or right upper quandrant)**
3. Management
   a. **Definitive treatment is delivery (obtain immediate obstetrics consult).**
   b. **Until that can be accomplished, give drug therapy. (Do not do a pelvic examination or transport the patient before drug therapy.)**
      (1) **Magnesium sulfate**
         (a) **Has both anticonvulsant and antihypertensive properties that usually control seizure activity but may not be adequate for blood pressure control; diazepam and/or hydralazine may also be needed.**
         (b) **Loading dose is 4–6 g IV over 5–15 minutes; then continuous IV infusion at 1–3 g/hr; patient should be placed on a cardiac monitor and have a Foley catheter in place.**
         (c) **Excretion is 100% renal, so urine output should be maintained at a rate of ~25 mL/hr.**
         (d) **Stop maintenance infusion if deep tendon reflexes disappear.**
         (e) **Watch for signs of magnesium toxicity**
            i. **Respiratory depression**
            ii. **Bradydysrhythmias**
            iii. **Loss of deep tendon reflexes**
         (f) **Antidote: calcium gluconate (1 g slow IV push)**

(2) If seizure continues, use typical anticonvulsant drugs: benzodiazepines (eg, lorazepam up to 0.1 mg/kg IV or diazepam 5 mg/min IV every 5 minutes as needed up to 20–30 mg)

(3) Phenytoin (10 mg/kg IV over 1 min) or diazepam (5 mg/min IV every 5 minutes as needed up to 20–30 mg) may be given for seizures resistant to magnesium therapy. If phenytoin is used, the potential for toxicity is reduced by giving this lower-than-normal loading dose, because serum phenytoin levels tend to be higher in pregnant patients (due to hypoalbuminemia); diazepam can produce both maternal and fetal depression but is a good choice for prolonged seizures (or status epilepticus) resistant to magnesium therapy.

(4) Hydralazine is indicated for the treatment of hypertension associated with eclampsia that does not improve with administration of magnesium sulfate. Its use is generally restricted to patients who continue to have a diastolic blood pressure >110 mmHg. Dosage is 5–10 mg IV slowly every 20 minutes as needed to keep the diastolic blood pressure between 90 and 100 mmHg.

(5) Labetalol is also safe and effective in controlling hypertension in eclamptic patients. It should be used when hydralazine is ineffective or unavailable. The initial dose is 20 mg IV and can be doubled every 10 minutes until blood pressure control is achieved or 300 mg has been given.

I. HELLP syndrome

1. A very severe form of preeclampsia (although some experts consider HELLP syndrome an independent disorder) characterized by:
   a. **H**emolysis
   b. **E**levated **L**iver enzymes and
   c. **L**ow **P**latelets (<100,000/mm^3)
2. Associated with increased maternal death
3. Clinical presentation
   a. Any symptoms associated with simple preeclampsia
   b. Right upper quadrant pain with nausea and vomiting are the most common.
   c. Commonly include generalized edema, right upper quadrant tenderness, jaundice, GI bleeding, and hematuria.
4. Management
   a. Depends on the severity of disease and the gestational age of the fetus. Delivery is the ultimate cure.
   b. Platelet transfusion should be pursued if the platelet count is <20,000/mm^3.

J. Rh immunoprophylaxis

1. Rh-negative mothers who are carrying Rh-positive babies and are exposed to any of the clinical events described below are at risk of developing Rh isoimmunization. This can negatively affect the outcome of both their current and subsequent pregnancies, producing fetal anemia, fetal hydrops, and fetal loss.
2. Events imposing a risk
   a. Miscarriage
   b. Ectopic pregnancy
   c. Term delivery
   d. Trauma

e. **Placenta previa**
f. **Abruptio placenta**
g. **Vaginal bleeding of unknown etiology**

3. **To prevent this, check the type and Rh of all pregnant women with any risk factors, and administer Rh immune globulin to those women who are Rh-negative and have not been sensitized in the past.**
4. **Dosing of Rh immune globulin**
   a. **A minidose of 50 mcg IM should be given for spontaneous abortions <12 weeks gestation; >12 weeks requires a standard dose of 300 mcg IM, possibly more (see Kleihauer-Betke test, page 607).**
   b. **Administer within 72 hours of the event.**

K. Premature rupture of membranes: the leading cause of endometritis and puerperal sepsis
1. Definition: rupture of fetal membranes before onset of labor
2. Clinical presentation: history of watery vaginal discharge in small or large amounts
3. Diagnostic evaluation: after ultrasound to confirm placental placement if bleeding
   a. Perform a careful sterile speculum examination without using a lubricant (which can give a false-positive), and obtain sterile swabs of the posterior vaginal vault for:
      (1) A nitrazine test: amniotic fluid is alkaline (blood and semen give false-positives) and turns the yellow paper to blue-green.
      (2) Microscopic examination of a dried slide: amniotic fluid should show "fern" (blood and inflammation give false-negatives).
   b. Obtain cervical cultures for *N gonorrhoeae, Chlamydia,* group B streptococci, aerobes, and anaerobes for patients with preterm (occurring before 37 weeks gestation) rupture of membranes. A digital examination should not be done in suspected or confirmed cases.
4. Management is hospitalization.
   a. If gestational age is >37 weeks → delivery within 12–24 hours
   b. If gestational age <37 weeks → time of delivery depends on fetal lung maturity and development of infection (chorioamnionitis)
5. Complications: these patients are subject to development of chorioamnionitis.

L. Postpartum (puerperal) fever
1. Etiology: look for all usual causes of fever
   a. Wound infection (cesarean or episiotomy)
   b. Urinary tract infection (especially if there is a history of catheterization or forceps delivery)
   c. Pneumonia
   d. Phlebitis
2. Endometritis
   a. Clinical presentation: swollen, tender uterus with foul lochia 1–3 days after birth
   b. Management: hospitalization for broad-spectrum antibiotics IV
3. Pelvic thrombophlebitis
   a. May mimic endometritis; suspect this diagnosis if patient does not respond quickly to antibiotics.
   b. Can cause pulmonary embolus

M. Mastitis

1. Etiology
   a. Usually caused by *Staphylococcus aureus*
   b. Usually occurs as a complication of breast-feeding (but can occur antepartum and in postpartum women who are not breastfeeding)
   c. Usual mode of transmission is inoculation of breast milk from the infant's nasopharyngeal secretions; cracked nipples can also be a route of entry for organisms.
2. Clinical presentation
   a. Cellulitis involving one segment of a breast (initial infection)
   b. Abscess formation (may result if the initial infection is untreated)
   c. Painful swollen breast
   d. Fever (often high)
   e. No pus expressed from the nipple (unless there is an abscess close to it)
3. Diagnostic evaluation: Gram stain of expressed milk often reveals *S aureus*
4. Management
   a. Simple mastitis
      (1) Warm compresses and analgesics for symptomatic relief
      (2) Breast-feeding should be continued (as long as the bacteria is not antibiotic-resistant), because this assures continued drainage; the infant will not be harmed because he or she is already colonized.
      (3) Oral antibiotics (penicillinase-resistant, eg, dicloxacillin or a cephalosporin)
   b. Mastitis with abscess formation
      (1) Incision and drainage should be referred to a surgeon (for drainage in the operating room).
      (2) Continued drainage with a breast pump is indicated, because higher concentrations of bacteria may be harmful to the infant; some authors recommend discontinuation of breast feeding until the infection has cleared.

IV. SURGICAL COMPLICATIONS OF PREGNANCY

A. Ectopic pregnancy

1. **Leading cause of maternal death in the first trimester and the second leading cause (overall) of maternal death**
2. **Usually presents 5–8 weeks after the last normal menses**
3. **Risk factors (~50% of ectopic pregnancies have no associated risk factors)**
   a. **High risk**
      (1) **Previous ectopic pregnancy, tubal surgery, or sterilization procedure**
      (2) **Documented tubal scarring/pathology**
      (3) **Diethylstilbestrol exposure in utero**
      (4) **Presence of intrauterine device (prevents only intrauterine (not extrauterine) pregnancies)**
   b. **Moderate risk**
      (1) **History of pelvic inflammatory disease**

(2) Infertility (in vitro)
(3) Multiple sex partners

c. Low risk
(1) Previous pelvic/abdominal surgery
(2) Cigarette smoking
(3) Vaginal douching
(4) Age of first intercourse <18 years old

4. Clinical presentation

a. Patients classically present with a history of amenorrhea followed by abdominal pain and abnormal vaginal bleeding.
(1) Classic triad is present in only 15% of patients and is neither sensitive nor specific.
(2) The menstrual history may, in fact, be "normal," but pay attention to any subtle deviations; the onset, character, location, and intensity of the pain is quite variable and not particularly helpful.

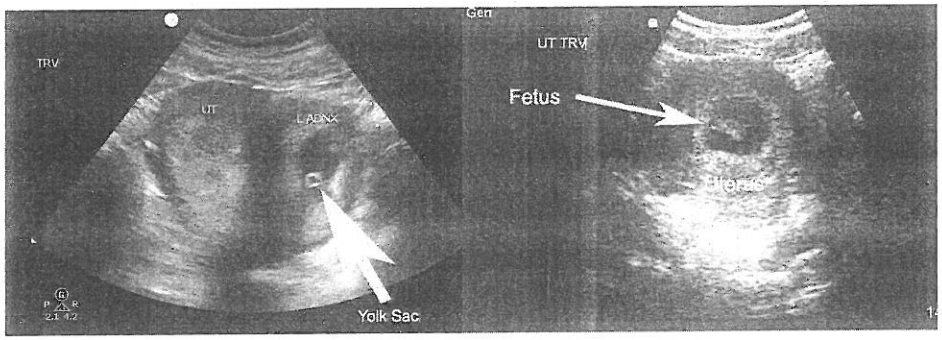
b. However, a clinical clue (if present) is shoulder pain referred from an irritated diaphragm (Kehr sign). Only 20% of ectopic pregnancies present in extremis.

c. Pelvic examination findings
(1) Vaginal bleeding (may be absent)
(2) Unilateral adnexal tenderness ± a palpable mass
(3) Uterus may be normal in size or slightly enlarged
(4) Fullness of the cul-de-sac

Table 24: Symptoms and Signs of an Ectopic Pregnancy

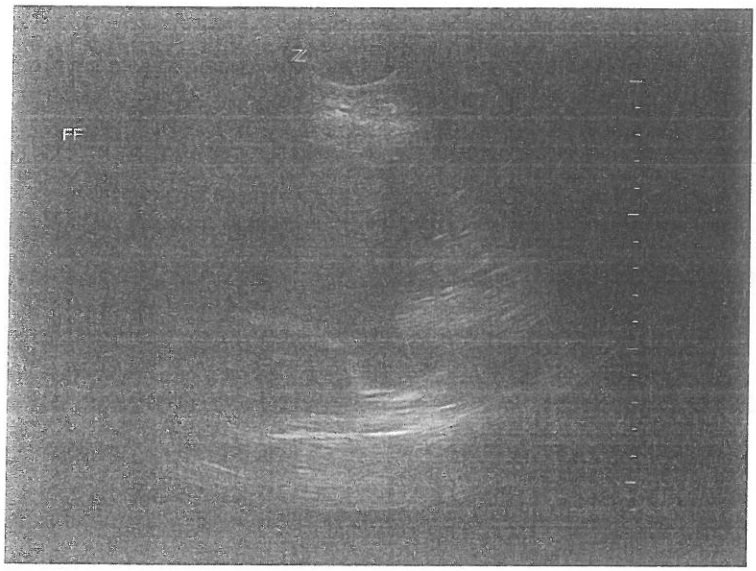
Symptom	% of Women with Symptoms/Signs
Abdominal pain	80–100
Amenorrhea	75–95
Vaginal bleeding	50–80
Dizziness	20–35
Pregnancy symptoms	10–25
Urge to defecate	5–15
Passing tissue	5–10
Sign	
Adnexal tenderness	75–95
Abdominal tenderness	80–95
Adnexal mass	50
Uterine enlargement	20–30
Orthostatic changes	10–15
Fever	5–10

5. Differential diagnosis
   a. Pelvic inflammatory disease
   b. Appendicitis
   c. Adnexal torsion
   d. Dysfunctional uterine bleeding
   e. Corpus luteum cyst
   f. Spontaneous abortion (complete or incomplete)
   g. Mittelschmerz
6. Diagnostic evaluation
   a. Pregnancy tests: a benign course cannot be assumed with low ß-HCG levels; ruptured ectopic pregnancies that require surgery have been reported with very low or absent levels of ß-HCG. The value of progesterone levels has yet to be clarified.
   b. Pelvic ultrasound
      (1) Transabdominal sonography is most useful in establishing the presence of an intrauterine pregnancy and excluding an ectopic pregnancy based on the low risk of heterotopic pregnancy in women who are not undergoing treatment for infertility. Visualization of a yolk sac (within the gestational sac) is the earliest reliable sign of an intrauterine pregnancy.
      (2) Transvaginal ultrasonography is an extremely sensitive technique; it can identify landmarks consistent with a normal intrauterine pregnancy at 5 weeks of gestation and may occasionally identify the actual ectopic pregnancy.
      (3) Indeterminate ultrasounds (positive ß-HCG but no definite intrauterine pregnancy or extrauterine findings of ectopic pregnancy) occur in up to 20% of emergency department patients undergoing evaluation for first-trimester emergencies; <30% of these go on to normal intrauterine pregnancies.
      (4) For patients in whom a ruptured ectopic pregnancy is a concern, check the right upper quadrant view/Morison's pouch for free fluid.



Courtesy of Sandra Werner, MD, RDMS, FACEP

Left: Empty uterus with a yolk sac in the left adnexa (ectopic pregnancy)
Right: Normal intrauterine pregnancy with a fetus surrounded by the uterus



Courtesy of Sarah Sommerkamp, MD
Free fluid separating liver, kidney, and bowel in a patient with a ruptured ectopic pregnancy

c. Management of vaginal bleeding or pain in the stable first-trimester pregnant patient
   (1) Sonography (transvaginal or transabdominal); must do transvaginal if transabdominal is indeterminate.
   (2) Ectopic pregnancy
      (a) Assess for stability.
      (b) If unstable FAST or right upper quadrant ultrasound for rupture
      (c) Obtain quantitative ß-HCG.
      (d) Consult obstetrics.
      (e) Consider methotrexate therapy versus surgery.
   (3) Indeterminate
      (a) Obtain quantitative ß-HCG.
         i. ß-HCG >1,500: ultrasound, serial ß-HCG measurements to establish ectopic pregnancy or miscarriage
         ii. ß-HCG <1,500: ultrasound, consult obstetrics, if stable do serial ß-HCG measurements, repeat ultrasound in 2 days or when ß-HCG is >3,000 for transvaginal ultrasound.
      (b) Assess clinical acuity.
   (4) Intrauterine pregnancy
      (a) Follow up with obstetrics.
      (b) Threatened miscarriage precautions
d. Culdocentesis
   (1) Consider for the unstable patient and in situations when ultrasonography is not available in a timely fashion.
   (2) Aspiration of nonclotting blood (a "positive" culdocentesis) is an indication for surgery.
   (3) A dry tap does *not* exclude the diagnosis.

7. Management

a. Establish an IV line and provide fluid resuscitation as indicated.
b. Obtain CBC, Rh typing as well as type and screen/crossmatch early in suspected cases.
c. Administer Rh immune globulin to women who are Rh-negative.
d. Definitive therapy
(1) Stable patients with unruptured ectopic pregnancies measuring <4 cm by ultrasound and ß-HCG <5,000 mIU/mL are eligible for methotrexate therapy.
(2) Stable patients with unruptured (or minimally ruptured) ectopic pregnancies measuring >4 cm by ultrasound → laparoscopic salpingectomy
(3) Unstable patients (or those in whom a laparoscopic approach is difficult) → laparotomy

B. Vaginal bleeding in pregnancy
1. First trimester bleeding
a. Before 12 weeks, vaginal bleeding is due to one of the following:
(1) Nonpregnancy-related conditions
(a) Infections
(b) Trauma
(c) Neoplasms
(2) Ectopic pregnancy
(3) Abortion
(4) Molar pregnancy
(5) Implantation bleeding
b. Threatened abortion is the most common cause of bleeding in a primigravida.
(1) Definition: any amount of bleeding without passage of products of conception or cervical dilatation
(2) Incidence: 30%–40% of clinically pregnant women have first trimester bleeding; 50% of these women ultimately miscarry.
(3) Management
(a) Bed rest for 24 hours
(b) No douching, tampons, or intercourse until bleeding resolves
(c) Rh immune globulin for Rh-negative women
(d) Follow-up with obstetrician in 2–3 days
c. Inevitable abortion
(1) Definition: the cervical os is open and vaginal bleeding is present.
(2) Management: observation, dilation and evacuation, or dilatation and curettage; Rh immune globulin for Rh-negative women. Consult obstetrics for brisk bleeding.
d. Incomplete abortion
(1) Definition: the cervical os is open, vaginal bleeding is present, and products of conception are present at the cervical os or in the vaginal canal.
(2) Management
(a) Visible products of conception should be removed with a ring forceps to control bleeding.
(b) Observation, dilation and evacuation, or dilatation and curettage; Rh immune globulin for Rh-negative women. Consult obstetrics for brisk bleeding.

e. Complete abortion
   (1) Definition: all products of conception have been passed, the cervix is closed, the uterus is firm and nontender, and the bleeding has almost stopped.
   (2) Management: up to 80% of first trimester miscarriages complete without intervention. Observation, curettage, or D&C (unless the patient has brought in all material she has passed which, on inspection, appears to be all products of conception, or complete expulsion has been confirmed by ultrasound); Rh immune globulin for Rh-negative women.

f. Missed abortion (more commonly called first or second trimester fetal demise)
   (1) Definition: failure to pass products of conception beyond 2 months after fetal death; pregnancy test converts from positive to negative.
   (2) Management: D&C; Rh immune globulin for Rh-negative women.

2. Late first trimester or second trimester bleeding → molar pregnancy
   a. Clinical presentation
      (1) Abdominal pain with severe nausea and vomiting (ß-HCG levels higher than normal)
      (2) Preeclampsia before 24 weeks
      (3) Vaginal passage of characteristic grapelike clusters of vesicles
      (4) Uterus large for dates
      (5) Anemia
   b. Diagnostic evaluation
      (1) Ultrasound (findings are typically described as a "snow-storm" appearance)
      (2) Discovery of molar tissue
   c. Management
      (1) D&C plus careful follow-up of ß-HCG titers to exclude chorionic carcinoma
      (2) Rh immune globulin for Rh-negative women

3. Second trimester bleeding
   a. Vaginal bleeding in the second trimester is due to:
      (1) Nonpregnancy-related conditions (infection, trauma, neoplasm) or
      (2) Abortion
   b. If abortion was the cause of bleeding, treatment depends on the type of abortion.
      (1) A threatened abortion is treated the same as in the first trimester.
      (2) If an inevitable, incomplete, or missed abortion occurs, evacuation of the uterus is usually done by oxytocin-induced labor in the hospital.
      (3) After a complete abortion, obstetrics consult should be obtained to determine the need for D&C.
      (4) Rh immune globulin should be administered to Rh-negative women.

4. Third trimester bleeding (after 28 weeks gestation)
   a. Placenta previa is responsible for 20% of cases of third trimester bleeding.
      (1) Definition: implantation of the placenta in the lower uterine segment such that it covers all or part of the cervical os
      (2) Clinical presentation
         (a) Painless, bright red vaginal bleeding that is sometimes intermittent and progressively severe over a period of 1–2 weeks
         (b) On abdominal examination, the uterus is usually soft and nontender.

(3) Risk factors
  (a) Prior cesarean section
  (b) Grand multiparity
  (c) Previous placenta previa
  (d) Multiple gestations
  (e) Multiple induced abortions
  (f) Maternal age >40 years
(4) Management
  (a) Establish IV access, draw blood for type and crossmatch and preoperative laboratory studies, establish cardiac and fetal monitoring.
  (b) Call obstetrics consultant immediately if patient is unstable and order ultrasound for placental location.
  (c) Pelvic examination (digital and speculum)
    i. Should not be done unless ultrasound excludes placenta previa
    ii. Should be done in the operating room by the obstetrician under "double set-up" conditions in patients who are bleeding rapidly, in labor, or are hemodynamically unstable. In this manner, an immediate cesarean section can be rapidly performed if uncontrolled bleeding results.
  (d) Administer Rh immune globulin to Rh-negative women.

b. Abruptio placenta: 30% of cases of third trimester bleeding
(1) Definition
  (a) Premature separation of a normally implanted placenta from the uterine wall causing visible or hidden bleeding.
  (b) Significant unseen blood loss can occur before or without vaginal bleeding.
(2) Clinical presentation
  (a) Painful uterine bleeding
    i. Bleeding is typically light (but may be heavy) and is usually dark or clotted.
    ii. At times, only pain is present, and this can vary from mild to severe.
  (b) Uterine findings vary from soft and nontender to slightly tender to rock hard.
(3) Severity of placental separation
  (a) Grade 0 or 1 (up to 40% cases): slight bleeding, little or no uterine irritability; no fetal distress or coagulation factor consumption
  (b) Grade II (45% of cases): moderate vaginal bleeding or hidden maternal blood loss, increased uterine irritability, fetal distress, and coagulation changes
  (c) Grade III (~15% of cases): uterine tetanic contractions, hypotension, coagulopathy, and increased possibility of fetal death
(4) Risk factors
  (a) Hypertension (most common)
  (b) Smoking, chronic alcohol consumption
  (c) Cocaine use
  (d) Multiparity
  (e) Increased maternal age
  (f) Previous abruptio placenta
  (g) Abdominal trauma

(5) Management

(a) Establish IV access; draw blood for type and crossmatch, preoperative laboratory studies, and disseminated intravascular coagulation profile; establish cardiac and fetal monitoring.

(b) Fluid resuscitation and blood replacement as needed

(c) Obtain immediate obstetrics consult, and perform ultrasound to exclude placenta previa (ultrasound is not sensitive enough to exclude abruptio placenta).

(d) Stable patients may be observed in the hospital with close surveillance if a surgical team is available.

(e) If the patient is unstable → immediate delivery by cesarean section (unless patient is in advanced labor); place the patient in the left lateral decubitus position to relieve inferior vena caval compression (can increase cardiac output by 30%–40%).

(f) Administer Rh immune globulin to Rh-negative women.

c. Uterine rupture

(1) Causes in an unscarred uterus

(a) Oxytocin stimulation

(b) Cephalopelvic disproportion

(c) Grand multiparity

(d) Placenta percreta (abnormal adherence of the placenta to the uterine wall with invasion of the myometrium all the way to its peritoneal covering)

(e) Blunt abdominal trauma

(2) Causes in a scarred uterus

(a) Previous hysterotomy, myomectomy, cesarean section, or curettage

(b) Manual removal of placenta

5. Postpartum hemorrhage

a. Immediate postpartum hemorrhage

(1) Definition: blood loss >250 mL immediately after delivery or >500 mL within the first 24 hours after delivery

(2) Etiology

(a) Uterine atony (most common cause)

(b) Retained placental tissue

(c) Genital tract trauma (lacerations, uterine rupture, inversion)

(3) Clinical presentation/pelvic examination

(a) Palpate for a large, soft, atonic uterus.

(b) Manually locate and remove placenta if it does not pass.

(c) Look for and treat lacerations (pack or repair) and inversion.

(4) Management for uterine atony

(a) Bimanual massage (one hand on abdomen, one in vagina); if uterine contractions are not induced, suspect uterine rupture.

(b) IV oxytocin: add 10–40 units to 1 L of IV fluid, and run at a rate sufficient to control uterine atony (~10 mL/min).

(c) Methylprostaglandin $F_{2\alpha}$ 0.25 mg IM (or methylergonovine 0.2 mg IM) if bleeding persists despite above measures

b. Delayed postpartum hemorrhage
   (1) Definition: hemorrhage occurring >24 hours after delivery (most often 1–2 weeks after birth)
   (2) Etiology
      (a) Retained placental tissue (confirm with ultrasound)
      (b) Endometritis
   (3) Management
      (a) Control bleeding with oxytocin or methylergonovine.
      (b) Start antibiotics in patients with evidence of endometritis (fever, purulent discharge, and/or uterine tenderness).

C. Appendicitis in pregnancy
1. The most frequent surgical emergency of pregnancy
2. Incidence of appendicitis is not higher in pregnancy, but the development of complications (especially rupture) is more frequent in pregnant patients because of delay in diagnosis.
3. Clinical presentation
   a. After the first trimester, the diagnosis can be elusive, because the normal leukocytosis of pregnancy frequently does not increase further and the pain location changes (may gradually move up to the right flank or right upper quadrant).
   b. In the third trimester, appendicitis is most frequently misdiagnosed as pyelonephritis, because the appendix occupies a position in close proximity to the right kidney at this point in pregnancy and can thereby produce right flank pain and pyuria (without bacteriuria).
      (1) High fever, chills, bacteriuria, and pyuria must all be present before the diagnosis of pyelonephritis is certain.
      (2) If "pyelonephritis" has not improved with IV antibiotics in 24 hours and the patient is not afebrile in 48 hours, reconsider the diagnosis.
4. Management: while ultrasonography can be helpful in excluding other diagnoses, laparoscopy or laparotomy is the preferred diagnostic procedure.

D. Abdominal trauma in pregnancy
1. **Important points**
   a. **You have two patients, and maternal circulation will be maintained at the expense of the fetus.**
      (1) Trauma-induced maternal shock carries with it a fetal mortality rate of almost 80%.
      (2) During the initial evaluation, concentrate on the clinical status of the mother because fetal survival depends wholly on maternal integrity.
   b. **The patient should be kept on her left side at all times (except during the abdominal examination) to avoid a supine hypotensive syndrome caused by pressure of the gravid uterus on the inferior vena cava, which reduces venous return. Cardiac output can be augmented 30%–40% by placing the patient in the left lateral decubitus position.**
2. **Lactated Ringer's should be used for fluid resuscitation.**
3. **Factors contributing to the difficulty of evaluation**
   a. **Because of the physiologic anemia of pregnancy (↑ in plasma volume > ↑ in RBC volume), the normal hematocrit in late pregnancy is 31%–35%.**

b. Because of the increased plasma volume associated with pregnancy (40%–50% increase compared with the nonpregnant state), a pregnant patient can lose 30%–35% of her blood volume before demonstrating the usual signs of hypovolemic shock.

c. Blood pressure is decreased in the second trimester (usually <125/75 mmHg); however, a systolic blood pressure <80 mmHg is always abnormal.

d. The WBC count is increased in the second and third trimesters (as high as 18,000/mm^3).

e. Abdominal findings are altered by the presence of an enlarged uterus.

4. All pregnant women (>20 weeks gestation) who sustain abdominal trauma should undergo a minimum of 4 hours of cardiotocographic monitoring once their vital signs have been stabilized. Signs of fetal distress include:

   a. Fetal bradycardia or tachycardia

   b. Late decelerations (bradycardia after a contraction)

   c. Loss of fetal beat-to-beat variability: tocographic monitoring is important to evaluate uterine contractions

5. Fetomaternal hemorrhage

   a. Abdominal trauma during pregnancy results in fetomaternal hemorrhage in up to 30% of patients.

   b. If an Rh-negative woman carrying an Rh-positive fetus is undetected and untreated, isoimmunization can result.

   c. Thus, all Rh-negative women who sustain abdominal trauma during pregnancy should receive Rh immune globulin.

   d. Kleihauer-Betke test

      (1) Although the standard dose of 300 mcg is sufficient in most cases of gestation >12 weeks, the Kleihauer-Betke test can be helpful in quantifying the exact dose of Rh immune globulin to be given.

      (2) Should be used in pregnant women who have sustained abdominal trauma to detect and quantify the amount of fetomaternal hemorrhage that has occurred.

      (3) This information is useful in determining the amount of Rh immune globulin to administer, because fetomaternal hemorrhage >30 mL requires more than the standard dose.

      (4) It is only used in women who are at least 12 weeks pregnant, because the fetal volume of blood up until this point is <30 mL and, therefore, is covered by the standard dose of Rh immune globulin.

      (5) It is only useful when it is positive; a negative test does not exclude a small fetomaternal hemorrhage capable of sensitizing an Rh-negative mother.

6. Abruptio placenta is the number one cause of fetal death after blunt trauma.

7. In the evaluation of abdominal trauma in the pregnant patient when ultrasound is not available or is indeterminate, culdocentesis can be substituted for peritoneal lavage. This is an important point, because pregnancy is a relative contraindication for peritoneal lavage.

8. Admission criteria

   a. Significant blunt abdominal trauma in a stable patient carrying a fetus >20 weeks gestation

   b. Vaginal bleeding

   c. Uterine irritability

   d. Persistent abdominal pain, cramping, or tenderness

e. **Evidence of maternal hypovolemia**
f. **Fetal heart tones <120 or >160 per minute**
g. **Presence of late decelerations**
h. **Amniotic fluid leak**
i. **Abnormal uterine sonogram: evidence of fetal injury or suspicious retroplacental structure (possible hematoma)**

9. **Postmortem cesarean section and fetal survival: a reasonably good chance if gestational age is >28 weeks (fetal weight 1,000 g) and the interval between maternal death and delivery is <15 minutes; perimortem cesarean section is indicated for cardiac arrest patients who are 22–24 weight-for-gestational age (WGA) or if the uterus is at or above the umbilicus.**
10. **All victims of trauma during pregnancy should be screened for domestic violence because of the high incidence of such during gestation.**

V. COMPLICATED DELIVERIES

A. Dystocia (difficult labor)
1. Types of dystocia
   a. Pelvic: abnormalities of the bony pelvis that affect the presenting fetal part
   b. Uterine
      (1) Ineffective contractions (hypertonic)
      (2) Maternal inability to assist expulsion
   c. Fetal
      (1) Malpresentation (most common cause of fetal dystocia)
      (2) Multiple gestations
      (3) Large fetal size (secondary to gestational diabetes)
      (4) Congenital anomalies
2. Management
   a. Pelvic dystocia → cesarean section
   b. Uterine dystocia → treatment is based on the nature of the contractions
      (1) Hypotonic → oxytocin drip
      (2) Hypertonic → tocolysis
   c. Fetal dystocia →fetal manipulation: maneuvers for shoulder dystocia (head is already delivered)
      (1) Flex the mother's legs over her abdomen → rotates the pubic symphysis upward → allows greater exposure of posterior shoulder (McRobert maneuver), *or*
      (2) Rotate fetus into an oblique position and apply suprapubic (not fundal) pressure, *or*
      (3) Apply suprapubic pressure and gentle downward pressure on the fetal head, *or*
      (4) Reach into the vaginal canal and gently extract the posterior arm and shoulder—do not force this!

d. Episiotomy may be used in conjunction with above maneuvers. As a last resort, the Zavanelli maneuver (replacement of fetal head into pelvis, followed by cesarean section) and other extreme maneuvers such as clavicular fracture can be performed.

B. Nuchal cord

1. Umbilical cord wrapped around fetal neck; ideally reduced over infant's head after delivery of head and before delivery of shoulders.
2. If unable to reduce over head, may somersault infant out, keeping head close to perineum, or clamp and cut and proceed with expedited delivery.



Courtesy of Wesley Fox

C. Prolapsed cord

1. Types of umbilical cord prolapse
   a. Overt: cord lies below the presenting part and can be visualized or palpated
   b. Occult: cord lies adjacent to the presenting part and can be detected by fetal monitoring (fetal heart rate changes with intermittent cord compression)
2. Etiology
   a. Abnormal presentations (especially transverse)
   b. Cephalopelvic disproportion
   c. Multiple gestations
   d. Placenta previa
   e. Prematurity
   f. Premature rupture of membranes
3. Management
   a. Administer oxygen.
   b. Reposition the mother.
      (1) Overt cord prolapse → knee to chest position (apply continuous upward pressure on the presenting part to relieve pressure on the cord until the fetus is delivered by cesarean section)
      (2) Occult cord prolapse → lateral (or Trendelenburg) position
         (a) If fetal heart rate returns to normal with cord compression → vaginal delivery
         (b) Persistence of fetal heart rate change with cord compression → cesarean section

D. Breech delivery

1. Preferred delivery in North America is by cesarean section.
2. Occurs in 3%–4% of term pregnancies
3. Feared complication is head entrapment.
4. Keep hands off the breach until umbilicus delivers.
5. Deliver shoulders by rotation, position fingers on maxillary process to flex head.
6. If head entrapment, use terbutaline or nitroglycerin for uterine relaxation.

E. Uterine inversion

1. Traction on the umbilical cord during delivery can result in partial or complete uterine inversion.
2. Clinical presentation
   a. Partial inversion → crater-like depression over the suprapubic area and descended uterine fundus and cervix on vaginal examination
   b. Complete inversion → uterus is outside the body and associated with immediate life-threatening hemorrhage
3. Management
   a. Immediate anesthesiology and obstetrics consult
   b. Two large-bore IV lines with lactated Ringer's run wide open
   c. Type and crossmatch for 6–8 units of blood
   d. Tocolytic drugs (terbutaline, ritodrine, $MgSO_4$) to relax the uterus so that manual reimplantation can be achieved
   e. Try to reimplant the uterus *without removing the placenta* (↑ risk of hemorrhage). If this fails, do not proceed further until:
      (1) IV lines are secured and running well.
      (2) Blood is available.
      (3) Anesthesia is being administered to the patient.
   f. Gently separate the placenta from the uterus. Then place your hand on top of the fundus with fingers extended (so they touch the border of the cervix), and apply pressure to the fundus (along the axis of the vagina) to push the fundus back through the cervix (remember, the uterus is "inside out"—it is inverted through the cervix and can be reimplanted only by pushing it back again through the cervix). If manual reimplantation fails, emergency laparotomy is indicated.
   g. On completion of successful reimplantation
      (1) Discontinue tocolytic therapy.
      (2) Start oxytocin therapy (while manually maintaining the uterus in position so it does not "reinvert").
      (3) Administer broad-spectrum antibiotics.

F. Multiple births

1. Be suspicious of multiple gestations if the mother:
   a. Used "fertility drugs" before pregnancy *or*
   b. Gained more weight than she expected *or*
   c. Noticed more fetal movement recently
2. If the uterus is larger than normal for dates (and the history is suspicious), order an ultrasound to confirm the diagnosis.

3. Anticipate potential complications
   a. Spontaneous abortion
   b. Premature rupture of membranes, labor, infant
   c. Placenta previa
   d. Abnormal presentation
   e. Postpartum uterine atony
   f. Resuscitation of multiple babies

G. Stillbirth

1. Maternal risk factors (eg, excessive obesity, advanced age, illness, drug abuse) have been associated with stillbirth.
2. Etiology
   a. Congenital anomalies/polyhydramnios
   b. Placenta previa/abruptio
   c. Multiple gestations/breech presentations

H. Emergency perimortem cesarean section and fetal survival

1. **If the fetus is delivered within 5 minutes of maternal cardiac arrest (unassociated with prolonged hypoxia before arrest) and gestational age is >28 weeks, fetal survival is improved.**
2. **If the fetus is delivered >15 minutes after maternal cardiac arrest, fetal survival is poor.**
3. **Perimortem cesarean section is indicated for cardiac arrest patients who are 22–24 WGA or if the uterus is at or above the umbilicus.**

GYNECOLOGIC AND OBSTETRIC DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A 25-year-old man presents with a painless rash that has been on his penis for over a week. On examination, you see heaped-up edges surrounding a painless ulcer.

What is the diagnosis?

Scenario B

Presentation: A 23-year-old woman presents with copious amounts of gray vaginal discharge and a fishy odor. On physical examination, you see red spots on her cervix but no cervical motion tenderness.

What is the diagnosis?

Scenario C

Presentation: A sexually active young woman complains of bilateral lower abdominal pain for several days (often following menses) associated with an abnormal vaginal discharge. She has fever >100.4°F (°38C), chills, and general malaise.

Physical examination: On physical examination, there is lower abdominal tenderness, cervical motion tenderness, and bilateral adnexal tenderness.

What is the diagnosis?

Scenario D

Presentation: A 20-year-old woman presents with abrupt onset pain lateralized in the pelvis. The pain is 10/10 and started after intercourse. She has had no other symptoms, discharge, or fever.

What is the diagnosis?



Scenario E

Presentation: A 28-year-old pregnant woman at 32 WGA presents complaining of right-sided chest pain and shortness of breath. She is tachycardic and tachypneic and has a low oxygen saturation. Her legs are swollen, and she has just taken a long car trip.

What is the diagnosis?

Scenario F

Presentation: EMS arrives with a visibly gravid woman who is actively seizing.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: syphilis

Scenario B

Diagnosis: trichomoniasis

Scenario C

Diagnosis: pelvic inflammatory disease

Key facts:

- This classic clinical scenario is seen in only one-third of cases.
- Constitutional symptoms (fever >100.4°F [38°C], chills, and general malaise) are frequently present.
- GI complaints, such as nausea and vomiting, may also be present but are less common.

Scenario D

Diagnosis: adnexal torsion

Scenario E

Diagnosis: pulmonary embolism

Scenario F

Diagnosis: eclampsia (preeclampsia and grand mal seizures or coma)

